

NEUROMUSCULAR CONTROL OF THE HIP, PELVIS, AND TRUNK
DURING RUNNING

by

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A DISSERTATION

Presented to the Department of Human Physiology
and the Graduate School of the University of Oregon
in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy

September 2017

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Degree awarded September 2017

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DISSERTATION ABSTRACT

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Patellofemoral pain syndrome (PFPS) is the most common injury in runners and has a significant female sex bias. Current evidence suggests that several proximal factors, including hip muscle strength, hip muscle activation, and hip kinematics during running, play a large role in the development of PFPS, particularly in females. However, the relationships between these variables are unclear. A better understanding of these relationships in both males and females could help clinicians develop targeted interventions for this syndrome. Thus, this dissertation is comprised of four studies aimed to better understand the relationships between these risk factors.

The first study investigated whether there are any relationships between hip muscle strength and hip muscle activation during running. Overall, hip muscle strength and hip muscle activity during running do not appear to be strongly related.

The second study used a multiple regression approach to look for predictors of hip adduction and hip internal rotation during running. Sex was a significant predictor in both models, and running speed, static subtalar inversion range of motion, and gluteus maximus amplitude were significant predictors in the hip adduction model.

The third study examined the effect of decreasing hip abduction strength on running kinematics and hip muscle EMG. After the fatigue protocol, there were no

changes in gluteus medius amplitude or timing, and no changes in hip kinematics during running. However, there were some changes in kinematics, particularly at the trunk, as well as differences in gluteus maximus and tensor fascia latae activation.

Finally, the fourth study used an alternative biomechanical method called continuous relative phase (CRP) to investigate the effect of sex and decreasing hip abduction strength on CRP variability at the hip. Decreasing hip abduction strength increased frontal plane CRP variability from 20-40% of stance phase, primarily in females, and females demonstrated less CRP variability than males in the frontal plane and transverse planes.

Overall, the results from this study improve our understanding of the relationships between hip strength, hip muscle activation, and hip kinematics during running in both males and females, which may have implications for knee injury rehabilitation strategies.

This dissertation includes unpublished co-authored material.

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PUBLICATIONS:

Hannigan J, Osternig L, & Chou L-S (2017). Sex-Specific Relationships between Hip Strength and Hip, Pelvis, and Trunk Kinematics in Healthy Runners. *Journal of Applied Biomechanics*. In Review.

ACKNOWLEDGMENTS

I could not have been more fortunate for Dr. Li-Shan Chou to accept me into his lab five years ago. Without your superior mentorship, I would not be the scientist, researcher, or educator I am today. Over the past five years, you have allowed me the freedom to develop my own research ideas, while helping me construct these ideas into this dissertation project. You have devoted countless hours to my graduate progress – from teaching me kinetics, to reviewing my thesis and manuscript drafts, to providing continued professional guidance, your dedication to my success as a graduate student is not measurable in words.

I would like to thank the members of my dissertation committee – Drs. Louis Osternig, Stan James, Brian Dalton, and Ken Prehoda – for your critiques, suggestions, and guidance during my graduate education and dissertation progress. Dr. Osternig, your meticulous input on my thesis and dissertation writing has pushed me to analyze my data more critically, and pick my words more precisely. I will always fondly remember our times spent on the sidelines of Oregon football games together. Dr. James, this project would not have been possible without your clinical expertise. Thank you especially imparting you knowledge of knee injuries, and for your assistance in developing the clinical examination. Dr. Dalton, you spent an entire term teaching me 1-on-1 about EMG data collection and analysis, which helped immensely in developing this dissertation. Without this support, I would likely still be struggling to analyze the EMG data.

In addition to the members of my committee, I would like thank Drs. Andy Karduna, Anita Christie, Michael Hahn, and Robert Wayner. In working with you all

over the past five years, you have each left a lasting impact on my growth as an educator, scientist, and clinician.

I would like to express a heartfelt thank you to all of the members of the Motion Analysis Laboratory. First, thank you to Dr. Jim Becker, who, took me under his wing during my first graduate year and imparted his extraordinary knowledge of biomechanics and running injuries. Thank you to the graduate students, past and present - David Howell, Chi-Wei Chou, Amy Lo, Quinn Peterson, Teresa Chen, Will Pitt, and Frankie Qu – for your support, assistance, and camaraderie over the past five years. Also, thank you to the undergraduate and high school students who worked with me – Varneet Brar, Chris Gronseth, Justine Silberberg, Hao Tan, Colin Lipps, and Peter Shen – your hard work and dedication in helping to collect, analyze, and process data for this project was more helpful and valuable than you likely realize.

Most importantly, I would like to thank my family and friends. Mom and Dad – I could not be more blessed to have you as my parents, and I cannot possibly put into words how much your continued love and support over the past 28 years means to me. Every day while living in Oregon, I have looked forward to our nightly phone conversations. While this sounds clichéd, I would not be where I am today without you. Mary, you have tolerated my quirks while continuing to be a source of unwavering love, support, and of course, delicious food. We made it through two years apart, and I cannot wait to share new adventures together.

Finally, this project would not have been possible without the financial support from the Betty Foster McCue Fellowship for Human Performance and Development (UO Graduate School), the Marthe E. Smith Memorial Science Scholarship (UO College of

Arts & Sciences), and the Jan Broekhoff Graduate Scholarship (UO Department of Human Physiology).

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CHAPTER I

INTRODUCTION

Health Benefits and Risks of Running

According to the World Health Organization, approximately 68% of deaths each year are due to non-communicable chronic diseases such as cardiovascular disease, type 2 diabetes, cancer, and chronic respiratory disease (124). The five leading risk factors for these diseases are high blood pressure, tobacco use, high blood glucose, physical inactivity, and obesity (9). Physical inactivity itself has been linked to high blood pressure, high blood glucose, and obesity, meaning a low level of physical activity is either directly or indirectly related to four of the five top risk factors for chronic disease (9).

Running is a very popular method of physical activity. Regular running is associated with improved aerobic fitness, cardiovascular function, metabolic fitness, adiposity, and postural balance (83). In the United States alone as of 2013, almost 10 million people reported running at least 110 days per year, with an additional almost 20 million running between 25-109 days per year (73). This accounts for approximately 15-20% of the United States' population (33,73). From 2004 to 2013, running participation has increased by about 70% in the United States (73). While this increase in running participation may help combat the rise of chronic diseases (9), running comes with its own set of risks – namely, injury.

Depending on the competitive level of runners sampled and definition of injury, somewhere between 20 to 80% of runners will develop an injury over the course of a year

(38), meaning approximately 6 to 24 million runners are injured annually. Between 40-70% of injured runners seek medical treatment for these injuries (55,68), meaning that high medical costs are associated with these injuries. Many runners seek treatment because these injuries not only prevent them from participating in physical activity, but also interfere with activities of daily living and general quality of life (38).

Running injuries are predominantly multifactorial in nature (38,56). While studies vary slightly in how these risk factors are grouped, most studies agree that all injuries can be traced to some combination of training, systemic, health/lifestyle, and physiologic factors (12,38,49,77). While most factors within these subgroups are specific to a particular injury, a couple factors are common amongst all injuries: training errors and previous injuries.

Training errors, although arguably the most modifiable factor, appear to be the biggest culprit, responsible for up to 60% of all running injuries (56). These training errors include excessive mileage, intense workouts, a sudden change in training, and running on hills or hard surfaces (49,56,77). While most runners understand that training errors are a significant risk factor for injury, many admit they do not heed proper training precautions to prevent injury (94). Thus, encouraging runners to modify their training program should be an important emphasis for clinicians when releasing runners to resume their training after a setback (49,56).

Another very important risk factor, part of the health/lifestyle subgroup, is having sustained a previous injury, which has been shown to significantly increases one's susceptibility to incurring a subsequent injury (12,49,67). While resuming training too quickly may be a common factor in re-injury, another possibility is that one or more of

the underlying causes of injury was not addressed during rehabilitation (90). Many of these underlying causes fall into the physiologic category of factors, which includes muscle strength, flexibility and range of motion, muscle activation patterns during running, and running biomechanics.

Research on these physiologic factors has grown exponentially over the past 10 years, especially for injuries to the knee, which account for 40-50% of all running injuries (38,56,106). Of these knee injuries, patellofemoral pain syndrome (PFPS) has received the most attention, since it is considered the most common yet enigmatic running-related injury (106). Understanding the relationship between several risk factors for this injury serves as the backbone of this project, and will be discussed further in the next section.

Patellofemoral Pain (PFPS): Definition and Risk Factors

Definition and Prevalence

According to the 2016 Patellofemoral Pain Consensus Statement the core criterion in defining patellofemoral pain is “pain around or behind the patella, which is aggravated by at least one activity that loads the patellofemoral joint during weight bearing on a flexed knee” (17). Crepitus, tenderness, effusion, or pain during sitting can be present, but are not essential for a diagnosis of patellofemoral pain (17). Regardless, PFPS is the most common injury in runners, accounting for roughly 16-20% of all running-related injuries (106). It is a particularly important injury to study, as mounting evidence suggests that developing patellofemoral pain early in life increases the risk of developing patellofemoral osteoarthritis later in life (60,110,126). The injury also has a significant sex bias, as females are almost twice as likely to develop PFPS compared to males (106).

A General Proposed Mechanism of Injury

The exact mechanism for patellofemoral pain is not clear, as it is likely a multifactorial injury without one consistent mechanism (19). In general, the pain during running is thought to be caused by a combination of malalignment and/or maltracking (19), which can reduce the contact area between the patella and femur, increasing pressure in that area (91). Due to running's repetitive nature, this may overload either the subchondral bone, infrapatellar fat pad, peripatellar synovium, retinaculum, or patellar ligaments over time (19,36). When these repetitive stresses overload a tissue's ability to rebuild and recover, the structures where loads are the greatest may begin to degenerate, initiating a pain signal from joint mechanoreceptors (36). This pain appears greatest during squatting, stair ascent and descent, sitting, and running (25).

While this framework may help explain PFPS etiology broadly, the specific causes of malalignment or maltracking are only partially understood. Likely, these causes are a combination of local, distal, and proximal factors, which may vary between subgroups of PFPS sufferers (19).

Local Factors

Locally, the utility of the quadriceps angle, or Q-angle, has been controversial, as there is conflicting evidence that static Q-angle influences PFPS susceptibility. Pooled data from a 2013 meta-analysis, however, did in fact show that PFPS sufferers on average displayed a significantly larger Q-angle compared with controls (59). Greater lateral translation (24,122), lateral tilt (24,59,122), and hypermobility (123) of the patella may also predispose an individual to developing PFPS by affecting the normal alignment and arthrokinematics of the patellofemoral joint. However, these local factors can only

partially explain the causes of patellar maltracking (91), as the knee joint mechanics are largely influenced by factors both distal and proximal to the joint. Thus, both distal and proximal factors have been studied fairly extensively in relation to this injury.

Distal Factors

Despite extensive research, significant evidence pointing to distal factors is fairly limited. Starting at the foot, pooled meta-analysis data does not support a relationship between arch height and PFPS (74). In addition, although foot orthoses may help decrease knee pain during running (74), pooled data suggest there are no differences in peak rearfoot eversion between healthy and injured runners (22,75,80,81). One study did find greater shank internal rotation in the PFPS group compared to controls, but suggested that this finding may be linked to more proximal factors in their discussion (81). Thus, research over the past few 10 years has largely shifted to focusing on proximal factors, which have yielded more promising results.

Proximal Factors

Moderately strong retrospective evidence suggests that female runners with PFPS run with greater peak hip adduction (74,80–82,115,119) and peak hip internal rotation (81,82,101) compared to healthy female controls. Limited prospective evidence also indicated that females who subsequently developed PFPS ran with greater peak hip adduction at pre-injury baseline compared to females who did not sustain an injury (80). In addition to the hip, there is moderate evidence of greater contralateral pelvic drop (74,119) and knee varus (119) in males with patellofemoral pain, and weak evidence that females with PFPS may display greater contralateral trunk lean (81) compared to controls.

A major takeaway from these studies is that females and males with PFPS display markedly different proximal and local kinematics, which would appear to have opposite effects on the both the Q-angle and patellar tracking. Greater hip adduction, seen in female with PFPS, increases the Q-angle, and likely increases lateral patellar translation, which is thought to increase lateral patellofemoral joint stress (8,36). Conversely, increased knee varum, as seen in males, decreases the Q-angle, increasing medial patellar translation and likely medial patellofemoral joint stress (8,36). These findings are interesting, as females are already noted to run with greater hip adduction (31) and hip internal rotation (31,96) compared to males when healthy. These potential differences in etiology between sexes are noteworthy, especially considering the female sex bias of this injury. However, the lack of information on PFPS in males, particularly the void of prospective research, prevents any concrete conclusions.

In addition to kinematics, proximal muscle strength at the hip appears to be related to PFPS risk. Strong evidence from several retrospective studies has demonstrated that females with PFPS displayed significantly weaker hip abduction strength (11,23,52,71,93,101), hip external rotation strength (11,52,71,93), and hip extension strength (93,101) compared to healthy controls while injured. In addition, moderate evidence from retrospective studies suggests that females with PFPS displayed significantly weaker hip abduction strength (16,78,111), hip external rotation strength (16), and hip flexion strength (16,78) compared to the unaffected limb. While prospective research is limited, Finnoff et al. (34) did find that runners who developed PFPS displayed significantly weaker hip abductors and external rotators post-injury compared to their healthy pre-season measurements. It is important to note that a few

studies found no significant differences between injured runners and controls in regard to hip abduction and external rotator strength, both retrospectively (87) and prospectively (109). However, both of these studies measured muscle strength in testing positions that varied from standard procedures. As for males, no study has found a significant difference in hip strength between males with PFPS and healthy controls, lending more evidence to a sex-specific etiology for this injury.

Proximal hip muscle activation also may be related to PFPS injury risk. Willson et al. (117) found that females with PFPS displayed delayed and shorter duration gluteus medius activation compared to healthy controls. In addition, this study found that delayed gluteus medius and gluteus maximus onset was significantly correlated with greater hip adduction excursion, while delayed gluteus maximus activation was significantly correlated with greater hip internal rotation excursion (117). This finding thus appears to provide a link between the biomechanical and neuromuscular risk factors for this injury. In addition, studies have found that females with PFPS run with greater peak gluteus maximus activation (101) and rearfoot strikers with PFPS (mixed sex cohort) run with less peak gluteus medius activation (27) compared to controls.

Based on these aforementioned results, proximal factors for PFPS appear to be very important and worth further investigation. However, before exploring these factors further, it is important to discuss a different approach to running injury research that ignores individual risk factors and employs a more macroscopic technique.

Dynamical Systems Theory: Another Approach to Injury

In 1999, Dr. Joe Hamill published a paper exploring the use of dynamical systems theory in studying lower extremity overuse injuries (42). Since then, this approach has slowly been gaining traction in the running injury literature. Instead of studying individual injury factors, the dynamical systems accounts for the interaction of multiple injury factors by studying the variability of a system on a macroscopic level (42,44). A common quantitative method within this approach is called continuous relative phase, abbreviated CRP. This method combines both spatial and temporal information into one measure of coordination that can be used to quantify the variability of the system being studied – in this case, a runner (42,44).

The concept of coordinative variability is central to the dynamical systems approach, and needs to be distinguished from end-point variability. While end-point variability refers to variation in the end-goal of the motion (for example, hitting a bulls-eye in darts), coordinative variability refers to the manner in which the joints and segments involved in the motion interact to control movement (for example, how the shoulder, elbow, wrist and hand interact to throw the dart) (44). While most tasks demand low end-point variability, low coordinative variability has been implicated in overuse injury risk, as it may lead to repetitive loading of the same structures (42,44,69). This holds true for runners with PFPS, who appear to display lower CRP variability between the thigh and shank and between the shank and foot segment compared to healthy controls (42). However, proximal coordinative variability has not been studied in running, which appears important given the previously discussed proximal risk factors.

Dissertation Aims

While the dynamical systems approach adds to our understanding of PFPS etiology, its findings are hard to understand from a clinical perspective. Thus, while it's important to consider this approach, understanding individual risk factors for injury is still vitally important. For PFPS, while many biomechanical, neural, and muscular factors have been identified, the relationship between many of these factors is unclear, which complicates intervention strategies for this injury. In particular, while proximal muscle strengthening has become part of the standard of care for PFPS rehabilitation and has led to positive functional outcomes (1,10,26,30,32,45,57,74,84), proximal running kinematics do not appear to change as a result of a 6-8 week strengthening protocol (26,32,100,120,125). No studies to date have investigated whether proximal muscle strength is related to the amplitude or timing of proximal muscle activation during running. In addition, many risk factors for PFPS appear to sex-specific (119), which means that studying these relationships should consider sex as an important factor.

Therefore, the overall aim of this project is to examine the relationships between hip muscle strength, hip muscle activation during running, and running kinematics at the knee, hip, pelvis, and trunk in both male and female runners. To accomplish this overall aim, the project is divided into four main studies, each with a specific aim:

Aim 1. Hip muscle strength and hip muscle activation during running have both been cited as potential risk factors for PFPS. However, the relationship between these variables has not been studied. Therefore, the first specific aim is to determine the relationship between maximum hip muscle strength and hip muscle activation

(amplitude, onset, offset, and duration) during running. It was hypothesized that hip muscle strength and hip muscle activation will not be correlated.

Aim 2. Excessive hip adduction and hip internal rotation appear to place females at higher risk for developing PFPS. While some studies have looked at individual factors that may be correlated to hip adduction (117) and hip internal rotation (102,117), no study has attempted to look at multiple factors at the same time. Therefore, using a multiple regression approach, the second specific aim of this project is to determine whether any demographic, clinical, anthropometric, hip muscle strength, or hip muscle activation during running are related to hip adduction and hip internal rotation during running in both males and females. It was hypothesized that gluteus medius and gluteus maximus onset would be correlated with hip adduction excursion, and gluteus maximus onset would be correlated with hip internal rotation excursion in females only.

Aim 3. Proximal muscle strengthening over a 6-8 week period does not appear to alter proximal kinematics in healthy or injured runners. However, hip muscle activation during running may be related to hip kinematics. Thus, the relationship between hip strength, hip muscle activation, and proximal kinematics during running is still very unclear. It is important for clinicians to know how manipulating hip strength may change muscle activation and kinematics at the hip. However, strengthening has to be done slowly over time, meaning subjects may adapt and other factors can affect the results. Decreasing strength, however, can be done rapidly by fatiguing or weakening the muscle group of interest. Therefore, the third specific aim is to examine the effect of decreasing hip abduction strength on hip muscle activation (amplitude, onset, offset, duration) and

proximal running kinematics. It was hypothesized that decreasing hip abduction strength would not affect hip muscle activation or proximal running kinematics.

Aim 4. Low coordinative variability has been implicated in injury risk. No study has looked at the effect of decreasing hip abduction strength on CRP variability at the hip. Therefore, the fourth specific aim is to examine the effect of decreasing hip abduction strength on CRP variability at the hip and knee. It was hypothesized that CRP variability would increase with decreased hip abduction strength.

Flow of the Dissertation

This dissertation is structured in journal format and includes co-authored material written in preparation for publication in peer-reviewed journals. Following this introduction in Chapter I, Chapters II-V are individual studies to address each specific aim. Chapter II investigates the relationship between hip muscle strength and hip muscle activation during running and is co-authored by Drs. Li-Shan Chou and Brian Dalton. Chapter III then uses a multiple regression approach to investigate factors that may predict hip adduction and hip internal rotation during running and is co-authored by Drs. Li-Shan Chou, Brian Dalton, and Stan James. Chapter IV details the fatigue protocol methodology, as well as the effect of decreasing hip abduction strength on hip abductor muscle activity and proximal kinematics during running, and is co-authored by Dr. Li-Shan Chou. Chapter V investigates the effect of decreased hip abduction strength on CRP variability at the hip and is co-authored by Dr. Li-Shan Chou. Finally, Chapter VI provides a final discussion and conclusion, clinical implications, and future directions for research from the summative results of this dissertation.

CHAPTER II

THE RELATIONSHIP BETWEEN HIP MUSCLE STRENGTH AND HIP MUSCLE ACTIVATION DURING RUNNING IN MALES AND FEMALES

This chapter contains co-authored material. JJ Hannigan was responsible for the conceptual development, development of the protocol, data collection and analysis, and writing of the manuscript. Dr. Li-Shan Chou contributed to the conceptual development and refinement of the protocol, and provided critiques and editing for the manuscript. Dr. Brian Dalton provided technical advice for the EMG data collection and analysis.

Introduction

Weak hip muscle strength, particularly in the hip abductor muscles, appears to be a major risk factor for developing patellofemoral pain syndrome (PFPS) in females (11,23,52,71,93,101). Hip and core strengthening programs have been shown to increase strength while decreasing pain and improving functional outcomes in runners with PFPS (1,10,26,30,32,45,57,74,84). Such strengthening programs have thus become a cornerstone of PFPS treatment and rehabilitation.

One limitation of these programs, however, is that they do not appear to change lower extremity kinematics during running (26,32,100,120,125). Certain kinematic patterns during running, particularly “excessive” peak hip adduction and peak hip internal rotation, appear to place female runners at a greater risk of developing PFPS (74,80–82,101,115,119). Thus, if these strengthening programs do not change kinematics, these kinematic risk factors of injury remain present after rehabilitation, placing the runner at a high likelihood of re-injury.

Hip muscle activation during running may also play a role in the development of PFPS, possibly due to their relationship with hip kinematics. A study by Willson et al. (2011) demonstrated that females with PFPS display delayed and shorter gluteus medius activation compared to healthy females, which was significantly correlated with hip adduction excursion (i.e., as onset time became more delayed, hip adduction excursion increased). Excessive hip adduction excursion could lead to greater peak hip adduction, thus placing a runner at a higher risk of injury.

The relationship between hip strength and hip kinematics during running is not clear. While one study found significant correlations between isokinetic hip abduction strength and pelvic drop in a mixed-sex cohort (35) other studies have found no relationship between isometric hip abduction strength and hip adduction (4) or hip internal rotation (102) in females. Another study found that isokinetic hip abduction strength was correlated with hip adduction range of motion, but not hip internal rotation range of motion in males (107).

To help clarify this relationship, the dynamic activity of the hip muscles needs to be further studied in runners. No study has investigated whether isometric measures of muscle strength (as typically measured in-clinic) have any relationship between hip muscle activation during running. Knowing this relationship would help researchers and clinicians better understand the role of maximum muscle strength in PFPS injury risk.

Sex appears to be an important consideration when investigating these relationships, due to the sex differences risk factors for PFPS (119) and the previously cited relationships between strength and kinematics. Thus, the primary purpose of this study was to investigate the correlations between hip muscle strength and hip muscle

activity during running in males and females. A secondary purpose was to investigate sex differences in hip strength and hip muscle activity to compare to previous studies and add to the body of literature in this area. Based on this previous literature, it was hypothesized that hip muscle strength would not be related to any measure of hip muscle activity, but that females would display lower hip abduction and external rotation strength, and higher gluteus maximus amplitude compared to males.

Methods

Subjects

Prior to participation, all subjects signed an informed consent form approved by the University of Oregon (Appendix B). To be included in the study, subjects needed to be between 18-45 years old (81,101), average running at least 20 miles per week over the past month (129), and report no major injuries for at least the previous 6 months (6,21). A major injury was consistent with the consensus definition by Yamato et al. (127) as pain that required a restriction or stoppage of running for at least 1 week or 3 training sessions, or that required treatment from a medical professional (127).

Protocol and Equipment – Visit 1

Subjects visited the Motion Analysis Laboratory for two visits, separated by a minimum of 72 hours. Tests were performed on separate days so that performing the muscle strength assessment did not affect muscle recruitment patterns during running. The maximum isometric muscle strength assessment was performed during visit 1. Prior to performing the strength assessment, all subjects warmed up five minutes on the treadmill at their easy pace. All isometric muscle strength tests were then performed for

both limbs on the Biodex System 3 Dynamometer (Biodex Medical Systems, Shirley NY).

For all tests, the greater trochanter was aligned with the rotational axis of the dynamometer. Hip abduction strength was measured with the subject sidelying on a padded treatment table with the hip in 0-degrees of abduction, flexion, or rotation. The resistance pad was placed 3 finger lengths proximal to the joint line of the knee and secured tightly to the thigh (Figure 1A). Hip adduction strength was measured in the same position, except the hip was moved to 30° of abduction (Figure 1B).

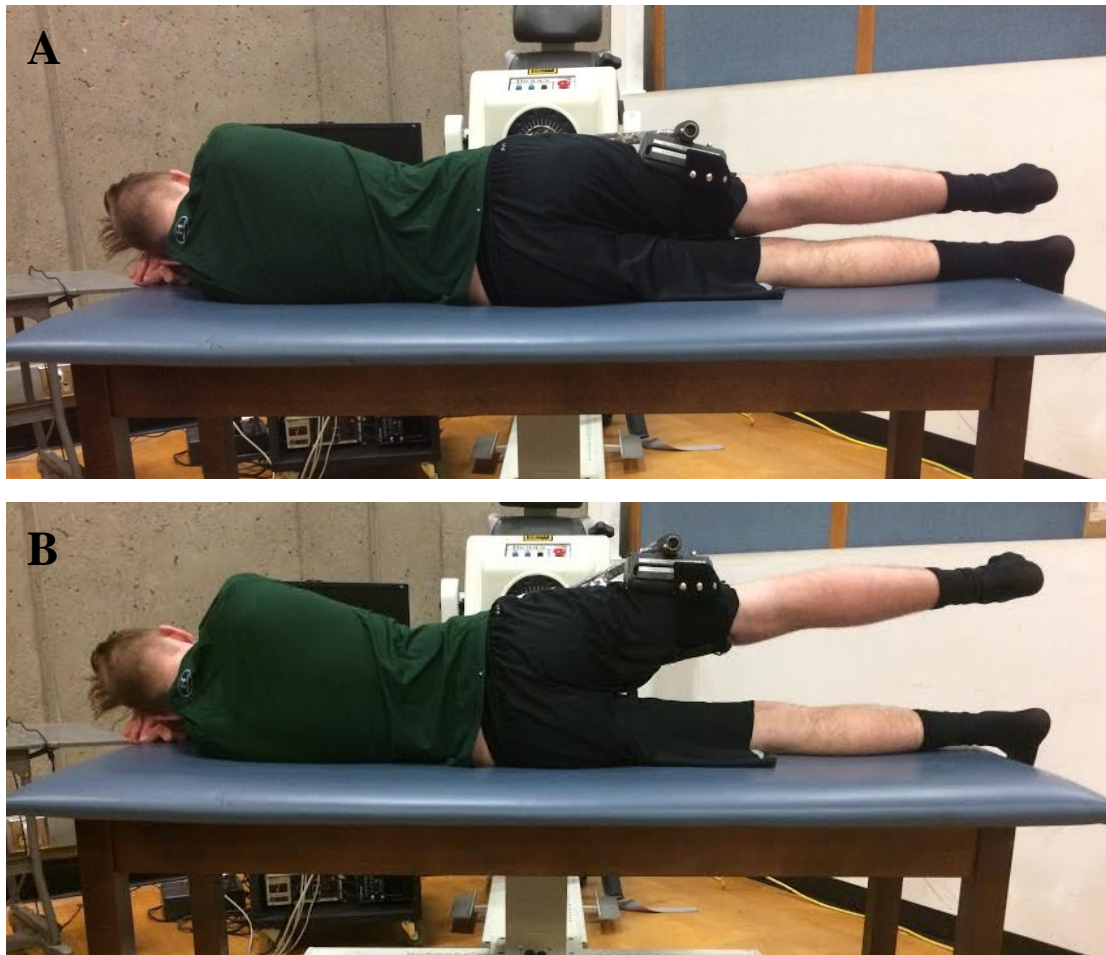


Figure 1. A) Hip abduction and B) hip adduction strength testing positions

Hip extension strength was measured with the subject prone on the treatment table with the hip in neutral and knee flexed to 90°. The resistance pad was placed 3 finger lengths proximal to the popliteal fossa and secured tightly to the thigh (Figure 2A). Hip flexion strength was measured with the subject supine on the treatment table with the hip flexed to 30°. The resistance pad was placed 3 finger lengths proximal to the superior border of the patella and secured tightly to the thigh (Figure 2B).



Figure 2. A) Hip extension and B) hip flexion strength testing positions

Hip internal and external rotation strength were both measured with the subject prone on the treatment table with the hip in neutral and the knee flexed to 90°. The

resistance pad was placed immediately proximal to the lateral malleolus and secured tightly to the distal shank (Figure 3).

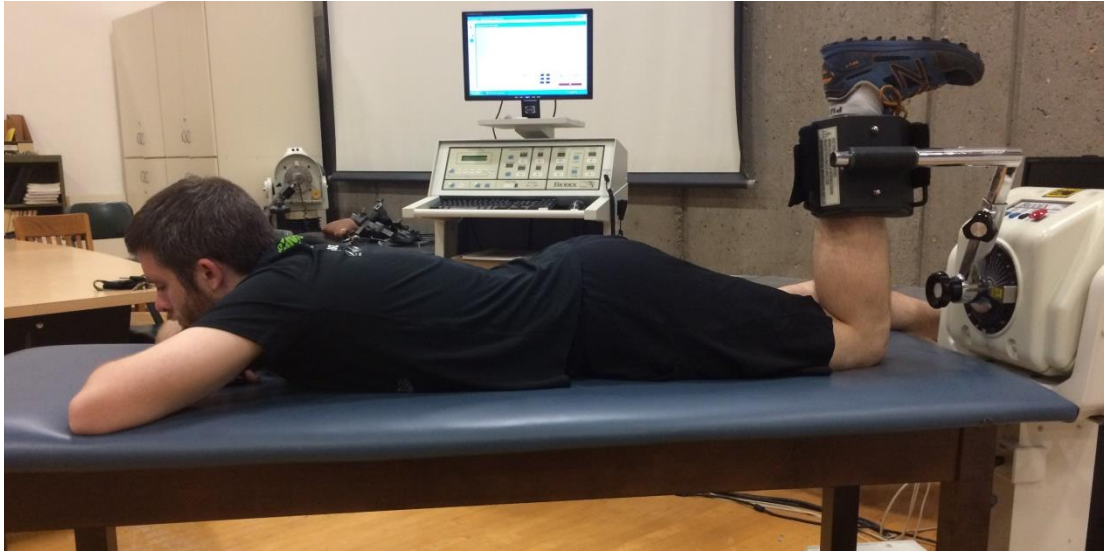


Figure 3. Hip internal rotation and external rotation strength testing positions

For each direction of movement, subjects pushed maximally three times for five seconds. Consistent verbal encouragement was provided for each maximal effort. For all tests except for hip internal and external rotation, these efforts were performed in a single set per limb with fifteen seconds in between efforts. Hip internal and external rotation strengths were alternated in the same set, with fifteen seconds in between each five-second effort. Subjects had at least one minute of rest between each direction of movement as the subject and dynamometer were repositioned.

Protocol and Equipment – Visit 2

During Visit 2, subjects began with a five-minute warmup at their easy pace on a treadmill. Subjects were then outfitted with a vest attached to a 6-channel EMG backpack with adjustable gain (MA-300, Motion Lab Systems, Baton Rouge LA) (Figure 4A).

Prior to electrode placement, the skin was lightly abraded and cleaned with an alcohol pad over the site of electrode placement locations. Six pre-amplified electrodes with 17mm inter-electrode distance (Figure 4B) and one ground electrode were then placed directly on the skin. Conducting gel was placed between the skin and electrodes and several strips of white athletic tape secured the electrodes to the skin. The active electrodes were placed on the tensor fascia latae (TFL), gluteus medius (GMED) and gluteus maximus (GMAX) bilaterally. The TFL electrode was placed vertically on the TFL muscle belly, located just inferior and posterior to the anterior superior iliac spine. The gluteus medius electrode was placed vertically in between the iliac crest and greater trochanter, approximately one-third of this distance below the iliac crest. The gluteus maximus electrode was placed on a line half the distance between the greater trochanter and the inferior lateral edge of the sacrum (116,117). The ground electrode was placed on the right clavicle. The positioning of electrodes can be seen in Figure 4C.

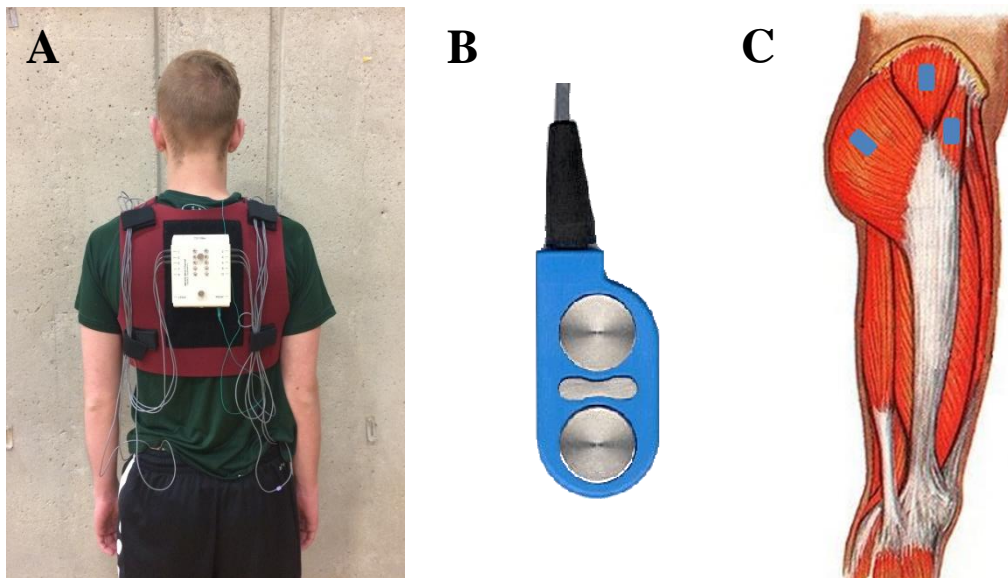


Figure 4. EMG Setup **A)** MA-300 backpack setup, **B)** MA-411 EMG pre-amplifier, **C)** Preamplified electrode placement on the TFL, GMED, and GMAX muscles bilaterally

The EMG pre-amplifier contained a band-pass filter from 15 to 3500 Hz and a gain of 20. At the backpack, the signal was band-pass filtered from 20 to 500 Hz, and the gain was manually adjusted to 4000 unless this produced signal clipping during the MVIC. This signal was then fed to a desktop amplifier (gain = 2) via a coaxial connection cable which fed the analog signal through a 12-bit A/D board connected to the motion capture computer in the Motion Analysis Laboratory. This signal was collected using Cortex 5 motion capture software (Motion Analysis Corporation, Santa Rosa CA) sampling at 1000 Hz.

The signal to noise ratio was visually inspected as the subject performed standing hip abduction and hip extension. If the signal to noise ratio was not acceptable, slight adjustments were made to the electrode placement and the signal to noise ratio was retested.

Maximum voluntary isometric contractions (MVICs) were then performed on a padded treatment table. For the gluteus maximus MVIC, the subject was positioned prone with the knee flexed to 90° (116,117). Two stabilization straps were tightly attached just distal to the gluteal fold and just proximal to the popliteal fossa on one leg. A researcher also helped stabilize the subject by placing their hands on the thigh during the test. Subjects were instructed to push up against the straps using their gluteal muscle with maximal force for five seconds. This was repeated for the same leg after 45 seconds and was then repeated on the opposite leg.

Based on pilot testing, MVICs for gluteus medius and TFL were performed with the subject sidelying and the hip in 0° of flexion and rotation, and 10° of abduction. Two stabilization straps were attached just proximal to the knee and just proximal to the ankle

with tautness that would allow 10° of abduction. Similar to GMAX MVIC testing, a researcher helped stabilize the subject's leg during testing to avoid the subject rotating their body. Subjects were instructed to push up against the straps using their hip abductor muscles with maximal force for five seconds. This was repeated for the same leg after 45 seconds and was then repeated on the opposite leg.

Subjects then ran continuous laps of approximately 40-meters in the Motion Analysis Laboratory at their easy run pace. This pace was defined as the pace a subject could comfortably maintain for 30 minutes while maintaining a conversation. Data were collected when the participants passed through a straight 10-meter region in the center of the capture volume. Participants were instructed not to alter their stride to hit three force plates (AMTI, Watertown, MA) located in series in this region. Subjects were given two laps to become accustomed to running in the lab before data were collected. Subjects then completed 20 laps in the laboratory.

Data Processing and Analysis

To quantify muscle strength, mean maximal torque for each strength parameter and limb was calculated by averaging the peak torque generated during the three efforts. This mean value was normalized by body mass for analysis.

Five running trials per limb per subject were selected for analysis (116,117). Only trials where the foot cleanly struck a force plate were selected. Foot strike was defined as the first frame where the vertical ground reaction force exceeded 50 Newtons (N), and toe off was defined as the first frame where the vertical ground reaction force fell below 50 N.

All EMG data were processed and analyzed using a custom LabView program. EMG data were band-pass filtered from 20-450 Hz, root-mean-square (RMS) averaged and smoothed using a 50 ms sliding window. To determine the MVIC for each muscle, both trials were processed. For both MVIC attempts, a sliding 500 ms window calculated the mean amplitude of the highest signal portion of the MVIC trial. The highest value between trials was determined the MVIC value for each muscle.

Running trials were trimmed to 250 ms before foot strike and 250 ms after toe off, respectively, for analysis. Due to variability in the data, no set threshold above baseline was determined feasible to consistently determine EMG onset. Therefore, EMG onset and offset was defined by visual inspection by the same examiner, which has shown to be reliable (53). EMG onset was defined as the first major vertical deflection of the EMG signal (53) during the 250 ms window before heel strike which remained above baseline for at least 25 ms (116,117). EMG offset was defined as the last major vertical deflection of the EMG signal (53) which remained below baseline for at least 25 ms (116,117). EMG onset is represented numerically as the time in milliseconds before foot strike, while EMG offset is represented numerically as the time in milliseconds before toe-off (Figure 5). EMG duration was calculated as the difference between the onset and offset times (Figure 5).

EMG peak amplitude was calculated as the maximum EMG recording within the visually determined onset and offset times (Figure 5). This maximum EMG value was normalized by the MVIC for that muscle. All four EMG parameters were then averaged between the five trials for analysis. In rare cases where the mean peak amplitude

exceeded 200% MVIC, this data point was discarded for analysis, as it was assumed the subject was not able to perform a true MVIC for that muscle.

Statistical Analysis

All EMG parameters were compared between sexes using unpaired *t*-tests. For all sex comparisons, the alpha-level was set to 0.05. Pearson correlation coefficients determined the relationship between strength and EMG measures. Due to the number of correlations performed, the alpha-level was lowered 0.01 to reduce the risk of committing a Type I error. All statistics were calculated using SPSS version 23 (SPSS Inc., Chicago IL), except for effect sizes, which were calculated using G*Power 3.1 (G*Power, Düsseldorf, Germany).

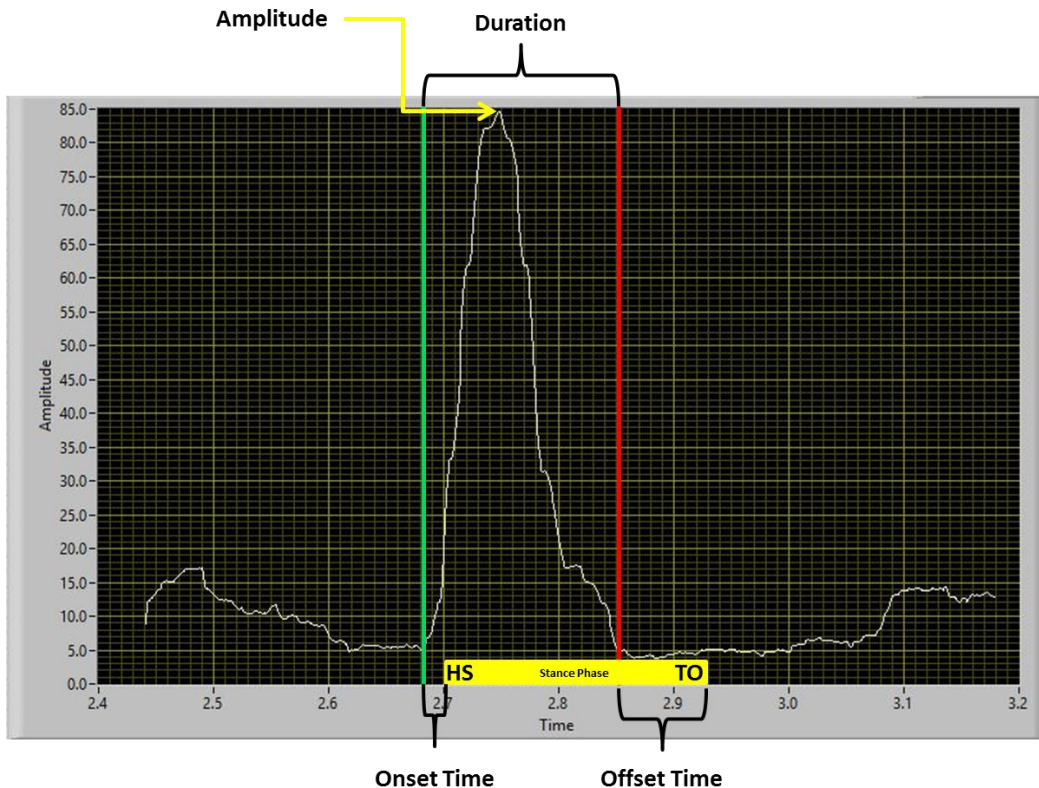


Figure 5. Sample visual depiction of EMG variable calculations. The green and red lines represent visually determined EMG onset and offset. The actual onset and offset variables used in this analysis were calculated relative to foot strike and toe-off. EMG duration was the time difference between visually determined EMG onset and offset. Peak amplitude was the maximum EMG signal between the EMG onset and offset.

Results

Thirty subjects, 15 males and 15 females, met the inclusion criteria and participated in the study. Subject demographics can be seen in Table 1.

Table 1. Subject demographics for females and males.

	Females	Males	<i>p</i> -value
Age (years)	27.5 ± 5.8	26.3 ± 8.3	0.648
Height (cm)	166.3 ± 4.3	177.8 ± 7.8	< 0.001*
Weight (kg)	59.1 ± 6.0	72.0 ± 11.4	< 0.001*
Weekly Mileage (miles)	27.7 ± 9.6	37.0 ± 15.8	0.061
Running Experience (years)	13.4 ± 5.7	11.5 ± 6.5	0.404
Running Speed (m/s)	3.0 ± 0.3	3.1 ± 0.4	0.300

* Indicates a significant difference between sexes, $p < .05$.

Males displayed significantly greater hip flexion strength ($p = .004$), hip internal rotation strength ($p = .025$), and hip external rotation strength ($p < .001$) compared to females. No differences were seen in hip abduction strength, hip adduction strength, or hip extension strength (Table 2).

Males displayed significantly earlier GMAX onset prior to foot strike ($p < .001$) and longer GMAX duration ($p = .019$) compared to females. There was a trend towards males displaying greater GMAX peak amplitude ($p = .061$) compared to females (Table 2).

Combining males and females together, there were no significant correlations between any measure of muscle strength and EMG. In males only, there were also no significant correlations between any measure of strength and EMG. In females, there was a significant correlation between hip abduction strength and TFL onset ($r = .494$, $p = .006$). No other correlations were significant (Tables 3 & 4).

Table 2. Sex differences in strength and EMG.

	Females	Males	<i>p</i> -value	Effect Size
Strength (Nm/kg)				
Hip Abduction	1.41 ± 0.26	1.51 ± 0.28	0.172	0.370
Hip Adduction	1.65 ± 0.34	1.82 ± 0.37	0.075	0.478
Hip Extension	1.20 ± 0.34	1.28 ± 0.33	0.360	0.239
Hip Flexion	1.04 ± 0.30	1.31 ± 0.38	0.004*	0.789
Hip Internal Rotation	0.36 ± 0.09	0.41 ± 0.08	0.025*	0.587
Hip External Rotation	0.30 ± 0.09	0.45 ± 0.10	< 0.001*	1.577
EMG (Amplitude = % MVIC; Onset, Offset, Duration = ms)				
TFL Amplitude	78.44 ± 40.03	97.06 ± 53.97	0.141	0.391
TFL Onset	44.35 ± 41.18	33.93 ± 30.97	0.281	0.286
TFL Offset	79.35 ± 20.79	76.76 ± 19.49	0.627	0.129
TFL Duration	214.58 ± 44.29	198.00 ± 34.75	0.118	0.417
GMED Amplitude	86.08 ± 30.42	82.97 ± 31.21	0.726	0.101
GMED Onset	25.47 ± 25.48	39.87 ± 47.44	0.150	0.378
GMED Offset	82.47 ± 24.91	73.67 ± 30.89	0.229	0.314
GMED Duration	196.03 ± 32.12	207.13 ± 46.09	0.284	0.279
GMAX Amplitude	84.08 ± 38.11	63.61 ± 38.12	0.061	0.537
GMAX Onset	24.17 ± 34.63	59.60 ± 30.62	< 0.001*	1.084
GMAX Offset	72.35 ± 23.76	72.43 ± 24.41	0.989	0.003
GMAX Duration	203.21 ± 40.71	228.00 ± 38.09	0.019*	0.629

* Indicates a significant difference between sexes, $p < .05$.

Table 3. Pearson correlation coefficients between hip strength and EMG measures.

	Hip Extension Strength		Hip External Rotation Strength	
	Females	Males	Females	Males
GMAX Amplitude	-0.027	-0.260	-0.295	-0.107
GMAX Onset	0.327	-0.082	0.013	-0.181
GMAX Offset	-0.010	0.285	-0.067	0.205
GMAX Duration	0.200	-0.188	-0.026	-0.150

Table 4. Pearson correlation coefficients between hip strength and EMG measures.

	Hip Abduction Strength		Hip Internal Rotation Strength	
	Females	Males	Females	Males
TFL Amplitude	0.022	-0.301	0.246	0.125
TFL Onset	0.494*	-0.142	0.329	0.017
TFL Offset	0.029	0.062	-0.108	0.066
TFL Duration	0.427	-0.178	0.252	-0.253
GMED Amplitude	0.014	-0.418	0.311	-0.104
GMED Onset	0.133	-0.094	0.034	-0.085
GMED Offset	-0.088	0.183	-0.026	0.159
GMED Duration	-0.115	-0.036	-0.022	-0.181

* Indicates a significant correlation, $p < .01$.

Discussion

The main purpose of this study was to investigate the correlations between measures of hip muscle strength and hip muscle activation during running. With one exception, there were no significant correlations between muscle strength and muscle activation parameters in either males or females, which mostly supported our hypothesis.

There were no significant relationships between maximum hip muscle strength and peak EMG amplitude. The three hip muscles we studied all displayed average peak amplitudes between 60 to 99% of MVIC during running in both males and females. These values are larger than may be expected, which could be due to the high degree of negative work, and thus eccentric muscle activity, noted for these muscles at the beginning of their activation prior to foot strike (65). However, despite peak EMG activity nearing MVIC levels during running, the maximum torque-generating capacity of the abductor and extensor muscle groups was not related to peak EMG amplitudes. This finding suggests that the relative amplitude of hip muscle activation for all three muscles is not dependent on the strength of the collective muscle groups they comprise. Thus, it

remains unclear what physiologic factors, if any, may modulate peak hip muscle EMG amplitudes during running.

One timing variable was related to maximum strength, as there was a significant, moderate, positive correlation between hip abduction strength and TFL onset time in females ($r = 0.494$, $p = .006$). In other words, as hip abduction strength increased, the time between TFL onset and initial foot contact also increased. While earlier muscle onset may help contribute to dynamic hip stability (117), this finding likely does not have significant implications on kinematics or injury rates, as the TFL appears to be a very minor contributor to hip stability during running (65).

We also saw a non-significant but trending, moderate, positive correlation between hip extension strength and gluteus maximus onset in females ($r = 0.327$, $p = .083$). While this relationship is non-significant, it is worth noting, as gluteus maximus onset has been negatively correlated to hip adduction and hip internal rotation excursion in females with PFPS (117). Thus, increasing hip extension strength may be considered during rehabilitation for females with PFPS.

All other timing variables were not correlated to any maximum muscle strength variables. Hence, it does not appear that maximum muscle strength has much relationship with the timing of muscle activation prior to foot strike, or the duration of hip muscle activity during running. The lack of relationship between hip abduction strength and gluteus medius onset and duration in females is particularly noteworthy, as lower hip abduction strength (11,23,52,71,93,101) and delayed gluteus medius activity (117) have been found in females with PFPS compared to controls. In addition there is limited evidence that suggests hip abduction strength (46) and gluteus medius onset (117) are

related to hip adduction excursion during running. However, the findings from this study suggest that these variables are not related, meaning that hip abduction strength and gluteus medius onset appear to be unrelated, independent risk factors for injury.

Males, on average, displayed significantly greater hip flexion, hip internal rotation, and hip external rotation strength compared to females. The hip external rotation strength finding supported our hypothesis. Greater hip flexion strength (95), hip internal rotation strength (95), and hip external rotation strength (63,95) in males compared to females has been found in other populations, but not exclusively runners.

Many (35,54,63,95,104), but not all (46) studies agree that healthy females have weaker hip abductors than males. This study agreed with the latter, as we did not see a significant difference between sexes, which did not support our hypothesis. While methodological differences may explain why our result was different than most, some studies used the same testing position and protocol as we did. The result from this study questions whether weaker hip abductor muscles can help explain the sex bias for developing PFPS, and indicates further research is needed.

Females displayed delayed and shorter GMAX activation compared to males. Willson et al. (2012) also found this trend, but their results did not reach statistical significance (116). Delayed gluteus maximus onset has been correlated to greater hip adduction and hip internal rotation excursion in females with PFPS (117). Since excessive hip adduction and hip internal rotation are both risk factors for developing PFPS, this finding may help explain the female sex bias for developing PFPS.

While our data just missed reaching statistical significance, we did see a trend towards females displaying greater gluteus maximus activation than males, which did

reach a moderate effect size ($p = .06$, $ES = 0.537$). Willson et al. (116) also found this result, which did reach statistical significance. While the significance of this finding is not entirely clear, Willson et al. hypothesized that relatively larger GMAX activation in females may lead to greater muscle fatigue and thus poorer dynamic control of the hip during prolonged running (116).

Also similar to Willson et al., we did not see any differences between males and females in gluteus medius timing or peak amplitude, suggesting that gluteus medius activation patterns do not contribute to the sex bias for developing PFPS (117). Gluteus medius timing differences during running have been cited between healthy and injured females, however, indicating that this parameter is still an important risk factor for injury (117).

Despite using different EMG systems and data processing steps, most EMG data presented in this study are very similar to Willson et al. (116) for both males and females. Comparing GMAX parameters between studies, average activation duration was within 12 ms for both males and females, while average peak amplitudes were within 4% of MVIC. For GMED, average activation duration was within 7 ms for both females and males. Average peak GMED amplitudes were slightly higher in Willson et al.'s study, with approximately the same mean difference between sexes. This difference is likely due to slight differences in MVIC testing position. While Willson et al. performed MVICs in 0° of abduction, we raised the hip to 10° of abduction, and saw average peak GMED amplitudes approximately 15% lower in both sexes. Thus, we recommend placing the hip in 10° of abduction for future studies, as it may yield slightly higher MVICs.

Overall, the degree of agreement in data between studies shows that our EMG measures are valid. Furthermore, similarities in EMG duration for both GMAX and GMED indicate that using visual inspection to determine onset and offset times is a reliable method in running studies.

There are a few limitations in this study. Surface EMG measures have several inherent limitations, such as muscle cross talk, movement artifact, and electrode placement errors, which can negatively affect the quality of EMG data. Foot strike patterns (i.e., rearfoot versus forefoot) were not controlled in this study. While foot strike pattern may affect distal muscle activation patterns (27,128), the effect on proximal muscle activation is unclear. Similarly, the type of running shoe was not controlled in this study, although no subjects wore minimalist or maximalist footwear. Finally, all subjects ran at the same relative effort, but not the same absolute pace. This was because we wanted to assess muscle activation patterns at a runner's easy pace, and assigning a specific pace would mean some runners may be running much faster or slower than normal. We did perform a post-hoc correlation between running speed and all EMG variables, and did not find a correlation between speed and onset, amplitude, or duration. There were also no differences in average running speed between males and females, so we do not believe that running speed affected any of the sex difference results.

In conclusion, hip muscle strength and hip muscle activity during running do not appear to be strongly correlated in healthy male or female runners. Differences in hip muscle strength and hip muscle activity between sexes, however, may have injury implications related to the sex bias for developing PFPS.

Bridge

Chapter II explored the relationship between hip muscle strength and hip muscle activation during running, which found these factors to be mostly unrelated. Chapter III uses these factors, along with clinical and anthropometric measurements, in a multiple regression model looking at predictors of hip adduction and hip internal rotation during running.

CHAPTER III

PREDICTORS OF HIP ADDUCTION AND HIP INTERNAL ROTATION DURING RUNNING

This chapter contains co-authored material. JJ Hannigan was responsible for the conceptual development, development of the protocol, data collection and analysis, and writing of the manuscript. Dr. Li-Shan Chou contributed to the conceptual development and refinement of the protocol, and provided critiques and editing for the manuscript. Dr. Brian Dalton provided technical advice for the EMG data collection and analysis. Dr. Stan James contributed to the development of the clinical examination.

Introduction

Current evidence suggests that females with patellofemoral pain syndrome (PFPS) run with greater peak hip adduction (74,80–82,115,119) and peak hip internal rotation (81,82,101) compared to healthy females. These biomechanical patterns are thought to decrease lateral patellofemoral contact area, which increases lateral patellofemoral contact stress (8,36). Over time, this increased stress may cause tissue degradation in the subchondral bone, infrapatellar fat pad, peripatellar synovium, retinaculum, or patellar ligaments, initiating a pain signal from mechanoreceptors (19,36).

While hip and core strengthening programs have yielded positive functional outcomes during PFPS rehabilitation (1,10,26,30,32,45,57,74,84), these programs do not appear to decrease hip adduction or hip internal rotation during running

(26,32,100,120,125). Thus, it appears that any reduction in knee pain stemming from these strengthening programs is not due to changes in hip or pelvis kinematics. This is problematic – if the biomechanical risk factors for injury are still present, this likely places the runners at a high risk of re-injury.

Gait retraining (5,51,72,74,79,118,121) and cadence modification (40,47,64) have been successful at altering hip kinematics during running. While these methods appear promising in altering running gait mechanics, factors outside of motor learning that may affect hip kinematics during running are still poorly understood, especially since the relationship between hip strength and hip kinematics appears tenuous (46). No study to date has investigated factors related to hip adduction during running. Factors affecting hip internal rotation during running have been marginally explored, as Souza et al. (2009) found hip extension endurance as a significant predictor of hip internal rotation (102). However, the number of predictor variables entered into their model was limited.

Risk factors related to demographics (59,106), anthropometry (24,59,60,122,123), static range of motion (59,74), hip strength (11,16,23,52,71,78,93,101,111), and hip muscle activation (74,117) have all been linked to knee injuries. Knowing which of these factors may be related hip adduction and hip internal rotation motion during running is important, especially for clinicians searching for targeted areas of intervention during rehabilitation. Therefore, the primary purpose of this study is to investigate factors related to hip adduction and hip internal rotation during running. It was hypothesized that sex and measures of hip muscle activation would be predictive factors for both biomechanical variables. Because of the hypothesized importance of sex in both models, a secondary purpose of the study was to investigate sex differences in the all variables

analyzed. Because sex differences in hip strength and hip EMG were already presented in Chapter II, sex differences in the clinical and kinematic variables will be presented here. It was hypothesized that females would display significantly greater static range of motion at the hip and greater hip adduction and internal rotation excursion during running compared to males.

Methods

Subjects

To be included in the study, subjects needed to be between 18-45 years old (81,101), average running at least 20 miles per week (129), and report no major injuries for at least the previous 6 months (6,21). A major injury was consistent with the consensus definition by Yamato et al. (2015) as pain that required a restriction or stoppage of running for at least 1 week or 3 consecutive days, or that required treatment from a medical professional (127). Prior to participation, all subjects signed an informed consent form approved by the University of Oregon (Appendix B).

Protocol and Equipment – Visit 1

Data collection was divided into two days separated by a minimum of 72 hours so that the muscle strength testing did not affect running kinematics. During day one, anthropometric, clinical, and strength data were collected after a five-minute warmup run. The clinical examination included measurements of range of motion, flexibility, and static posture, which are described in Table 5. All angle measurements were made with a goniometer by the same trained clinician, with the exception of standing Q-angle, which was calculated based on marker positions during the static motion capture trial.

Table 5. Description of clinical exam measures

Clinical Measure	Description
Standing Leg Varus to Floor (°)	Angle of the shank relative to vertical when standing barefoot with legs shoulder width apart
Standing Q-angle (°)	Angle between a line formed from the tibial tubercle to the patella and a line formed from the patella to the ASIS when standing with the legs shoulder width apart
Standing Arch Type	Clinician's assessment of standing arch type, ranging from 1 (very flat feet) to 5 (very high arch)
Arch Height Index	Truncated foot length (heel to 1 st MPJ) divided by height of the dorsal aspect of the foot measured at ½ of total foot length
Stork Test	Clinician's assessment of the movement (normal or restricted) of the PSIS relative to the sacrum as the subject balances on one leg and raises the other leg to 90° of hip flexion and knee flexion
Static Ankle Dorsiflexion (°)	Angle formed between the shank and foot during active ankle dorsiflexion with the knee extended (AD _f) and flexed (AD _e)
Static Ankle Plantarflexion (°)	Angle formed between the shank and foot during active ankle plantarflexion with the knee flexed
Static Subtalar Inversion (°)	Angle formed between the shank and foot during active ankle inversion in a prone position
Static Subtalar Eversion (°)	Angle formed between the shank and foot during active ankle eversion in a prone position
Static 1 st MPJ Dorsiflexion (°)	Angle formed between the great toe and the foot during active great toe dorsiflexion
Static Hip Internal Rotation (°)	Angle formed between the shank and horizontal as the thigh is internally rotated in a prone position and knee flexed to 90°
Static Hip External Rotation (°)	Angle formed between the shank and horizontal as the thigh is externally rotated in a prone position and knee flexed to 90°
Quadriceps Flexibility (°)	Angle formed between thigh and shank during maximum passive knee flexion in a prone position
Hamstring Flexibility (°)	Angle formed between the shank and vertical as the knee is passively flexed in a prone position with the hip flexed to 90°
Thomas Test (°)	Angle formed between the thigh and horizontal as the subject leans their trunk prone on a treatment table and lets their thigh
Ober's Test	Does the thigh fall below horizontal (negative) or stay above horizontal (positive) as the leg is extended and knee slightly bent while lying in a sidelying position
Seated Tibial Torsion (°)	Angle formed between the thigh and foot as the subject sits with legs off a table

All isometric muscle strength tests were then performed for both limbs on the Biodex System 3 Dynamometer (Biodex Medical Systems, Shirley NY). For each test, the greater trochanter was aligned with the rotational axis of the dynamometer. Strength testing positions can be seen in Chapter II (Figures 1-3). For each direction of movement, subjects pushed maximally three times for five seconds. Consistent verbal encouragement was provided for each maximal effort. For all tests except for hip internal and external rotation, these efforts were performed in a single set per limb with fifteen

seconds in between efforts. Hip internal and external rotation strengths were alternated in the same set, with fifteen seconds in between each five-second effort. Subjects had at least one minute of rest between each direction of movement as the subject and dynamometer were repositioned.

Protocol and Equipment – Visit 2

Subjects warmed up at their easy pace on a treadmill for five minutes and were then outfitted with a vest attached to a 6-channel EMG backpack with adjustable gain (MA-300, Motion Lab Systems, Baton Rouge LA) (Figure 4A).

Prior to electrode placement, the skin was lightly abraded and cleaned with an alcohol pad over the site of electrode placement locations. Six pre-amplified electrodes (Figure 4B) with 17mm inter-electrode distance and one ground electrode were then placed directly on the skin. Conducting gel was placed between the skin and electrodes and several strips of white athletic tape secured the electrodes to the skin. The active electrodes were placed on the tensor fascia latae (TFL), gluteus medius (GMED) and gluteus maximus (GMAX) bilaterally. The positioning of electrodes can be seen in Figure 4C.

The EMG pre-amplifier contained a band-pass filter from 15 to 3500 Hz and a gain of 20. At the backpack, the signal was band-pass filtered from 20 to 500 Hz, and the gain was manually adjusted to 4000 unless this produced signal clipping during the MVIC. This signal was then fed to a desktop amplifier (gain = 2) via a coaxial connection cable which fed the analog signal through a 12-bit A/D board connected to the motion capture computer in the Motion Analysis Laboratory. This signal was collected at 1000 Hz using

Cortex 5 motion capture software (Motion Analysis Corporation, Santa Rosa CA) so that EMG and motion capture data would be synchronized.

The signal to noise ratio was visually inspected as the subject performed standing hip abduction and hip extension. If the signal to noise ratio was not acceptable, slight adjustments were made to the electrode placement and the signal to noise ratio was retested.

Maximum voluntary isometric contractions (MVICs) were then performed on a padded treatment table. For the gluteus maximus MVIC, the subject was positioned prone with the knee flexed to 90° (116,117). Two stabilization straps were tightly attached just distal to the gluteal fold and just proximal to the popliteal fossa on one leg. A researcher also helped stabilize the subject by placing their hands on the thigh during the test. Subjects were instructed to push up against the straps using their gluteal muscle with maximal force for five seconds. This was repeated for the same leg after 45 seconds and was then repeated on the opposite leg.

Based on pilot testing, MVICs for gluteus medius and TFL were performed with the subject sidelying and the hip in 0° of flexion and rotation, and 10° of abduction. Two stabilization straps were attached just proximal to the knee and just proximal to the ankle with tautness that would allow 10° of abduction. Similar to GMAX MVIC testing, a researcher helped stabilize their leg during testing to avoid the subject rotating their body. Subjects were instructed to push up against the straps using their hip abductor muscles with maximal force for five seconds. This was repeated for the same leg after 45 seconds and was then repeated on the opposite leg.

Subjects were then outfitted with 39 reflective markers (6,41). The pelvis was defined by two markers on the anterior superior iliac spines, and one at the midpoint between the posterior superior iliac spines. The femur was defined by two markers at the medial and lateral femoral epicondyles, and one marker mid-thigh in line with the lateral epicondyle and greater trochanter. The hip joint center was defined by measurements of ASIS breadth (112).

Subjects then ran continuous laps of approximately 40-meters in the Motion Analysis Laboratory at their easy run pace while whole body kinematics were collected with a 10-camera motion capture system (Motion Analysis Corp., Santa Rosa CA) sampling at 200 Hz. This pace was defined as the pace a subject could comfortably maintain for 30 minutes while maintaining a conversation. Participants were instructed not to alter their stride to hit three force plates (AMTI, Watertown, MA) located in series in this region. Subjects were given two laps to become accustomed to running in the lab before data were collected. Subjects then completed 20 laps in the laboratory. Data were collected when the participants passed through a straight 10-meter region in the center of the capture volume.

Data Processing and Analysis

To quantify muscle strength, mean maximal torque for each strength parameter and limb was calculated by averaging the peak torque generated during the three efforts. This mean value was normalized by body mass for analysis.

Five running trials per limb per subject were selected for analysis (116,117). Only trials where the foot cleanly struck a force plate were selected. Foot strike was defined as the first frame where the vertical ground reaction force exceeded 50 Newtons

(N), and toe off was defined as the first frame where the vertical ground reaction force fell below 50 N (14). Marker trajectories were identified using Cortex 5.0 motion capture software (Motion Analysis Corp., Santa Rosa CA) and were smoothed using a low-pass, fourth-order, zero-lag Butterworth filter with an 8 Hz cutoff. A custom LabView program (National Instruments, Austin TX) calculated hip angles during stance phase using a YXZ cardan angle rotation sequence. Angular excursion of the hip was defined as the difference between the angle at initial contact and the peak angle during stance phase (4). Angular excursion was selected as the biomechanical variable in this study because it is less sensitive to between-day variability in marker placement compared to peak angles (29), represents the time period during stance phase where the hip muscles must act to decelerate the center of mass and control excessive hip motion (102), and has been used in several similar studies (4,35,107).

All EMG data were processed and analyzed using a custom LabView program. EMG data were band-pass filtered from 20-450 Hz, root-mean-square (RMS) averaged and smoothed using a 50 ms sliding window. To determine the MVIC for each muscle, both trials were processed. For both MVIC attempts, a sliding 500 ms window calculated the mean amplitude of the highest signal portion of the MVIC trial. The highest value between trials was determined the MVIC value for each muscle.

Running trials were trimmed to 250 ms before foot strike and 250 ms after toe off, respectively, for analysis. Due to variability in the data, no set threshold above baseline was determined feasible to determine EMG onset. Therefore, EMG onset and offset was defined by visual inspection, which has shown to be reliable (53). EMG onset was defined as the first major vertical deflection of the EMG signal (53) during the 250 ms

window before heel strike which remained above baseline for at least 25 ms (116,117). EMG offset was defined as the last major vertical deflection of the EMG signal (53) which remained below baseline for at least 25 ms (116,117). EMG onset is represented numerically as the time in milliseconds before foot strike, while EMG offset is represented numerically as the time in milliseconds before toe-off (Figure 5). EMG duration was calculated as the difference between the onset and offset times (Figure 5).

EMG peak amplitude was calculated as the maximum EMG recording within the visually determined onset and offset times (Figure 5). This maximum EMG value was normalized by the MVIC for that muscle. All four EMG parameters were then averaged between the five trials for analysis. In rare cases where the mean peak amplitude exceeded 200% MVIC, this data point was discarded for analysis, as it was assumed the subject was not able to perform a true MVIC for that muscle.

Statistical Analysis

All demographic, anthropometric, clinical, strength, and EMG variables served as potential predictor variables in the multiple regression models. These variables were checked for assumptions of linearity, homoscedasticity, normality, and multicollinearity to assess whether the assumptions for performing a multiple regression analysis were violated. Linearity and homoscedasticity were checked by visually inspecting the scatterplots of the studentized residuals against the unstandardized predicted values both collectively and for each predictor variable. Multicollinearity was checked by assessing the variance inflation factors (VIFs).

Due to high multicollinearity between variables, a principal component analysis (PCA) was run on the predictor variables (except for EMG measures, due to the number

of missing data points). This was done in order to assess which variables were highly correlated and could be grouped into the same principal components. Linearity between variables ($r > 0.3$) was checked, and any variable that was not correlated to another variable above this threshold was not included in the PCA. Sampling adequacy was checked using Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy ($KMO > 0.5$) and Bartlett's Test of Sphericity before principle components were calculated.

To assess multicollinearity between EMG variables, bivariate correlations were calculated between variables, with a threshold of $r > 0.8$ indicating significant collinearity between variables.

Bivariate correlations (Pearson's correlation coefficients) were then calculated between all predictor variables and both hip adduction excursion and hip internal rotation excursion. Bivariate correlations between the predictor and kinematic variables were assessed, with predictor variables of significance level $p < .10$ selected for inclusion in a stepwise multiple regression for each kinematic variable. If two or more variables within the same principle component fit this criterion, only one was selected for inclusion in the stepwise regression. Entry and exit criteria for the stepwise regression were set at $p < .05$ and $p > .10$, respectively. After performing both stepwise regressions, all assumptions of regression were rechecked.

Sex differences in clinical and kinematic measures were assessed using unpaired t -tests with an alpha-level of .05. All statistics were calculated using SPSS version 23 (SPSS Inc., Chicago IL), except for effect sizes, which were calculated using G*Power 3.1 (G*Power, Düsseldorf, Germany).

Results

Thirty subjects (15 males and 15 females) met the inclusion criteria and were included in this study. Subject demographics can be seen in Chapter II (Table 1).

All regression assumptions of linearity, homoscedasticity, and normality were tenable. However, numerous variables violated the assumption of multicollinearity, with many VIF values far exceeding 10. Thus, a principal component analysis was performed on all predictor variables except measures of EMG. EMG was excluded because the number of missing EMG data points exceeded the threshold to include in the PCA. In total, all four TFL parameters were not included for 1 male subject, GMED amplitude was not included for 4 subjects (3 male, 1 female), and GMAX amplitude was not included for 4 subjects (2 male, 2 female) due to poor data quality.

Subtalar inversion was removed from the PCA because it was not correlated with any other variable at the $r > .30$ level. The KMO measure of sampling adequacy was 0.502, which is considered just above the minimum threshold of 0.50 for performing a PCA. Bartlett's test of sphericity was statistically significant ($p < .001$), indicating that our data was suitable for PCA.

The first 6 principle components are listed in Table 6. All measures of hip strength were contained in PC 1. Descriptive variables of sex, height, and weight composed PC 2, along with hamstring flexibility. PC 3 contained measures at the thigh, along with 1st MPJ dorsiflexion. PC 4 contained static ankle range of motion measures. Age and running history composed PC 5, and leg varus and tibial torsion made up PC 6.

Table 6. Principal components and their percent of variance explained

PC	Percent of Variance	Variables
PC 1	16.7%	Hip Abduction Strength, Hip Adductor Strength, Hip Extension Strength, Hip Flexion Strength, Hip External Rotation Strength, Hip Internal Rotation Strength
PC 2	13.9%	Sex, Height, Weight, Hamstring Flexibility
PC 3	7.9%	Static Hip Internal Rotation, Quadriceps Flexibility, Standing Q-Angle, Static 1 st MPJ Dorsiflexion
PC 4	7.8%	Static Ankle Dorsiflexion (knee flexed), Static Ankle Dorsiflexion (knee extended), Static Ankle Eversion
PC 5	6.2%	Age, Running History
PC 6	5.8%	Leg Varus, Tibial Torsion

For all three EMG measures, onset and duration were significantly correlated at $r > 0.8$ and were thus considered multicollinear (TFL onset and TFL duration: $r = .870$; GMED onset and GMED duration: $r = 0.830$; GMAX onset and GMAX duration: $r = 0.821$).

Sex, height, weight, running speed, TFL duration, GMAX amplitude, arch type, static subtalar inversion, quadriceps flexibility, hamstring flexibility, and Ober's test were correlated with hip adduction excursion at $p < 0.10$. Height and weight were excluded in favor of sex due to their collective presence in PC 2. While hamstring flexibility was also included in PC 2, it was allowed into the stepwise regression as we determined its presence in this PC was likely due to the large sex difference in this variable (Table 11). The stepwise multiple regression model indicated that sex, running speed, GMAX amplitude, and subtalar inversion were all significant predictors of hip adduction excursion ($r = 0.633$, $r^2 = 0.401$) (Table 7-8). The assumption of multicollinearity was tenable in this final model (Table 8).

Table 7. Final variables added into stepwise hip adduction excursion regression model

Variable Added	<i>r</i>	<i>r</i>²	<i>r</i>² change	<i>F</i> change	<i>Sig F</i> change
Subtalar Inversion	0.365	0.133	0.133	7.219	0.010*
GMAX Amplitude	0.500	0.250	0.117	7.181	0.010*
Speed	0.575	0.330	0.080	5.365	0.025*
Sex	0.633	0.401	0.071	5.192	0.028*

*Indicates significant F change, $p < .05$

Table 8. Results of final regression model for hip adduction excursion

Variable	<i>B</i>	<i>St. Error</i>	<i>p-value</i>	<i>VIF</i>
(Constant)	-10.55	3.262	0.002*	
Subtalar Inversion	0.152	0.050	0.004*	1.007
GMAX Amplitude	0.015	0.007	0.048*	1.087
Speed	2.602	0.947	0.009*	1.035
Sex	1.278	0.561	0.028*	1.104

*Indicates significance in the final model, $p < .05$

Sex, height, weight, TFL offset, GMED onset, GMAX amplitude, GMAX offset, and external rotation strength were correlated with hip internal rotation excursion at $p < 0.10$. Height and weight were excluded in favor of sex due to their collective presence in PC 2. The stepwise multiple regression model indicated that only sex was a significant predictor of hip internal rotation excursion ($r = 0.351$, $r^2 = 0.123$) (Tables 9-10). The assumption of multicollinearity was tenable in this final model (Table 10).

Table 9. Final variables added into stepwise hip internal rotation excursion regression model

Variable Added	<i>r</i>	<i>r</i>²	<i>r</i>² change	<i>F</i> change	<i>Sig F</i> change
Sex	0.351	0.123	0.123	6.609	0.013

*Indicates significant F change, $p < .05$

Table 10. Results of final regression model for hip adduction excursion regression model.

Variable	<i>B</i>	<i>St. Error</i>	<i>p</i>-value	<i>VIF</i>
(Constant)	2.811	1.512	0.069	
Sex	2.460	0.957	0.013*	1.000

*Indicates significance in final model, $p < .05$

Sex differences in kinematic and clinical variables can be seen in Table 11. Females demonstrated greater hip adduction excursion and greater hip internal rotation excursion compared to males, $p < .05$. Females also demonstrated greater static ankle plantarflexion, static hip internal rotation, and hamstring flexibility, and less tibial torsion compared to males, $p < .05$.

Discussion

The main purpose of this study was to investigate significant predictors of hip adduction and hip internal rotation excursion during running. Stepwise multiple regression models indicated that sex, running speed, GMAX amplitude, and static ankle inversion range of motion were significant predictors of hip adduction excursion, and only sex was a significant predictor of hip internal rotation excursion. These findings partially supported our hypothesis.

Table 11. Sex differences in kinematic and clinical variables

	Females	Males	<i>p</i> -value	Effect Size
Kinematic Variables				
Hip Add Excursion	4.60 ± 1.84	3.30 ± 3.26	0.027*	0.491
Hip IR Excursion	7.73 ± 3.41	5.27 ± 3.26	0.006*	0.737
Clinical Variables				
Leg Varus	2.57 ± 3.55	2.20 ± 3.19	0.675	0.110
Ankle Dorsiflexion (ext)	5.03 ± 3.08	4.27 ± 2.74	0.313	0.261
Ankle Dorsiflexion (flex)	11.93 ± 3.46	10.77 ± 5.13	0.306	0.265
Ankle Plantarflexion	62.30 ± 8.09	54.13 ± 10.0	0.001*	0.898
Subtalar Inversion	23.00 ± 5.88	23.47 ± 4.93	0.740	0.087
Subtalar Eversion	9.46 ± 3.38	10.13 ± 5.13	0.555	0.154
1 st MPJ Dorsiflexion	45.30 ± 11.3	42.03 ± 15.4	0.353	0.242
Prone Hip IR	39.70 ± 9.58	34.23 ± 9.39	0.030*	0.577
Prone Hip ER	35.33 ± 8.86	33.63 ± 7.52	0.426	0.207
Quadriceps Flexibility	139.90 ± 9.80	139.20 ± 12.7	0.812	0.062
Hamstrings Flexibility	20.57 ± 10.5	33.57 ± 9.29	< 0.001*	1.311
Thomas Test	9.53 ± 9.21	9.67 ± 9.24	0.956	0.015
Tibial Torsion	4.53 ± 4.42	7.63 ± 4.69	0.011*	0.680
Arch Height	3.01 ± 0.24	3.02 ± 0.38	0.884	0.031
Standing Q-Angle	15.85 ± 7.02	17.77 ± 6.43	0.276	0.285

*Indicates a significant difference between sexes, $p < .05$

Being female was a significant predictor of hip adduction excursion during running (Table 7), and females displayed significantly greater hip adduction excursion compared to males (Table 11). This result is not surprising, as previous studies indicated that healthy females run with greater hip adduction, on average, compared to males (31). While some authors have hypothesized this may be due to sex differences in hip and pelvis anatomy (31), no clinical or anthropometric variables at the hip, including standing Q-angle, were significant predictors of hip adduction in our model. It is certainly possible that structural variables, such as femoral anteversion or the ratio between pelvic

width and femur length (102), could be significant predictors, but were not included in our analysis.

In the final model for hip adduction, a one unit increase in GMAX amplitude was significantly associated with a 0.15 unit increase in hip adduction (Table 8). This finding may indicate that runners with excessive hip adduction activate gluteus maximus at a higher percent of the maximum in an attempt to control frontal plane hip motion. This could potentially indicate the GMAX amplitude during running is less of a “predictor” and more of a “result” of greater hip adduction excursion. Though controlling frontal plane motion is not the primary function of gluteus maximus, some evidence suggests that the superior portion of the gluteus maximus does contribute to frontal plane torque at the hip (98,99).

GMAX amplitude’s inclusion in the model is interesting given the trend towards greater gluteus maximus activation in females from Chapter II (Table 2), which has also been cited in previous literature (116). Based on the combined results of these studies, female sex, gluteus maximus activation, and hip adduction excursion all appear to be related, which is likely significant given that excessive hip adduction is a potential biomechanical risk factor for PFPS in females.

A one unit increase in static subtalar inversion range of motion was significantly associated with a 0.152 unit increase in hip adduction excursion (Table 8). Due to the lack of sex difference in this variable, this variable’s inclusion in the final model appears to be independent of sex. This finding is difficult to explain, as during running the hip adducts for approximately the first 50% of stance phase, which coincides with ankle eversion, not inversion. One hypothesis is that greater static ankle inversion range of

motion may allow runners to land in a more inverted position, giving them more total available eversion range of motion at the ankle during the first 50% of stance phase. Thus, greater available eversion range of motion could allow greater hip adduction excursion during running, given that these movements are likely coupled during the first half of stance phase (20). The relationship between these variables certainly warrants further investigation.

A one unit increase in running speed was significantly associated with a 2.602 unit increase in hip adduction excursion (Table 8). This was also not surprising given that many joint excursions increase with as speed increases (92). This finding is important, as it indicates that regardless of sex, faster running is associated with an increase in hip adduction excursion, which may have negative consequences related to patellofemoral contact pressure (50). Thus, this finding gives runners more reason to exercise caution when incorporating speed work into their training program.

Gluteus medius amplitude and timing parameters were not included in the model for hip adduction excursion, which disagrees from the findings of Willson et al. (117), although Willson's study looked at correlations in females with PFPS. Gluteus medius is thought to be a major contributor to frontal plane hip stability during running (91), but gluteus maximus amplitude appeared to be a bigger predictor of hip adduction excursion during running. More research is needed to explain this finding.

The only significant predictor variable in the hip internal rotation model was sex (Table 9), and females were found to run with greater hip internal rotation excursion compared to males (Table 11). This finding partially supports our hypothesis and agrees with previous literature (31,46). Based on the results of Souza et al. (2009), femoral

structure, while possibly related to PFPS incidence, does not appear to be related to hip internal rotation motion during running in females (102). The results from our study also did not find any structural or clinical variables related to hip internal rotation excursion in our final model. Using a bivariate approach, Willson et al. (2011) found that gluteus maximus onset was significantly correlated with hip internal rotation excursion (117). However, the results of our study did not find a significant relationship between gluteus maximus onset and hip internal rotation excursion, which disagrees with the results from Willson et al. (117). It is possible that this difference between studies could be due to differences in calculating EMG onset, as we used visual inspection, while Willson et al. used a five standard deviation threshold above resting baseline.

Females demonstrated greater static ankle plantarflexion, static hip internal rotation, and hamstring flexibility compared to males, which partially supported our hypothesis. Greater static hip internal rotation may be due to structural differences at the pelvis and femur between sexes (70,102). The differences in hamstring flexibility also agree with previous literature (113,114). Females demonstrating greater static ankle plantarflexion range of motion appears to be a novel finding, but likely is not related to injury risk. The greater amount of external tibial torsion found in males is also interesting, as excessive shank internal rotation may be a risk factor for PFPS (81). Whether tibial torsion and shank rotation during running are related, however, is unknown.

While approximately 40% of the variance in hip adduction was explained by the variables in this study, only around 12% of the variance in hip internal rotation was explained. The relatively low amount of variance explained in both models likely

suggests that dynamic control of the hip during running is a complex neurophysiological and biomechanical process that may be very runner-specific. Hip muscle strength had been hypothesized as a significant factor related to hip and pelvis motion in runners (35,46,48,107). However, strength was not significantly correlated to joint excursions in either model. This finding may help explain why hip strengthening during PFPS rehabilitation could not significantly alter hip kinematics during running (26,32,100,120,125).

Every possible variable related to hip joint excursion during running could not be included in this study. While running shoes were not standardized, the type of running shoe worn was not entered into the regression equation, which may limit the results. Further analyses could explore whether more variance is explained by using a more homogenous subgroup of runners (example: forefoot versus rearfoot strikers, or a tighter inclusion age range). In addition, other biomechanical variables during running, both distal and proximal to the hip, were not included in this analysis. Our sample size was also relatively low, just above the minimum cutoff to perform a PCA. More subjects may be needed to increase the power of our regression model.

In conclusion, sex, running speed, gluteus maximus activation, and static ankle inversion range of motion were significant predictors of hip adduction excursion, and sex was a significant predictor of hip internal rotation excursion. While the relatively low amount of variance explained in both models suggests that dynamic control may be a complex process, the results will hopefully provide some insight into factors related to two major biomechanical risk factors for injury.

Bridge

Chapter III investigated variables that may be predictive of hip adduction and hip internal rotation excursion during running. Hip muscle strength had previously been hypothesized as a significant factor related to hip and pelvis motion during running. However, this variable was not a significant predictor of hip adduction or hip internal rotation excursion in our model. To further investigate this finding, Chapter IV uses a hip abductor fatigue protocol in order to look at inter-subject changes in running kinematics after hip abduction strength is decreased.

CHAPTER IV

THE EFFECT OF DECREASED HIP ABDUCTION STRENGTH ON RUNNING KINEMATICS AND HIP MUSCLE ELECTROMYOGRAPHY

This chapter contains co-authored material. JJ Hannigan was responsible for the conceptual development, development of the protocol, data collection and analysis, and writing of the manuscript. Dr. Li-Shan Chou contributed to the conceptual development and refinement of the protocol, and provided critiques and editing for the manuscript.

Introduction

Most literature agrees that females with PFPS have weaker hip abductor muscles, (11,23,52,71,93,101), and greater hip adduction (74,80–82,115,119) and hip internal rotation (81,82,101) during running than healthy females. However, based on previous literature, as well as the results of Chapter III, any relationships between hip strength and hip kinematics appear tenuous at best (4,32,46,48,100,120,125). One limitation of studies investigating the hip strength-hip kinematics relationship in runners is that they are either cross-sectional, or investigate changes in kinematics over a 4-8 week period of strengthening, where subjects can slowly adapt to increasing strength, and measurement reliability decreases between visits (29).

While muscle strength cannot be substantially increased during a single visit, the maximum torque-generating capacity of a muscle can be decreased after fatigue, by inducing muscle damage, or both (37,61,76). Decreasing hip abduction strength appears to alter spatiotemporal parameters during walking (3), movement strategies during static

balance control (7,61,76), and knee joint moments during cutting, jumping, landing, and running (37,85). However, decreasing hip abduction strength does not appear to significantly affect frontal plane hip and knee biomechanics during walking (88) or hip kinematics during running (37).

One potential avenue to assimilate these findings is investigating how trunk control is modulated with changes to hip abduction strength. It has been suggested, but not yet explored, that compensatory movements at the trunk may help explain how knee joint moments are altered without changes to hip and knee kinematics (100). We also do not currently know whether fatiguing and inducing muscle damage in the hip abductor muscles will affect hip muscle activation during running. Thus, the primary purpose of this study was to investigate kinematic and electromyographic differences during running before and after a hip abductor fatigue protocol. It was hypothesized that only trunk kinematics would be affected by the fatigue protocol.

In addition, because of cited sex differences in PFPS injury rates (106), running kinematics (31,86), and hip muscle activation during running (116), the effect of sex appears to be an important consideration when investigating this relationship. Therefore, this analysis will investigate whether males or females respond differently to decreasing hip muscle strength.

Methods

Fatigue Protocol Validation

Before proceeding with the main study, the fatigue protocol used to decrease hip abduction strength needed to be validated. To be included in the fatigue protocol

validation study, subjects needed to be between 18-45 years old, recreationally active for at least 30 minutes three days per week, and report no major injuries over the past six months (6,21). Prior to participation, subjects signed an informed consent form approved by the Institutional Review Board at the University of Oregon.

For this protocol, subjects first warmed up for five minutes on the treadmill. Subjects were then positioned sidelying on the padded treatment table with the greater trochanter of the dominant leg aligned with the dynamometer arm and the resistance pad attached firmly to the thigh three finger lengths above the joint line. The dominant leg was defined as the leg a subject would kick a soccer ball for distance. Subjects then performed three maximum isometric efforts in the direction of abduction with the hip in neutral position, separated by five seconds. The mean peak torque between these efforts was considered the subject's baseline hip abduction strength.

Subjects were then given explicit instructions for the fatigue protocol, and researchers ensured subjects were comfortable with the protocol before proceeding. The fatigue protocol was a dynamic two-minute task, as the arm of the dynamometer rotated at 30° per second through a 30° range of motion, regardless of the subject's exerted torque. All subjects were instructed to push in the direction of abduction for two minutes while the dynamometer rotated through this range of motion, with specific instructions to try to resist the dynamometer as it rotated downward. Thus, when the dynamometer rotated upward, the hip abductor muscles were contracting concentrically, and when the dynamometer rotated downward, the hip abductor muscles were contracting eccentrically. Subjects were given visual feedback of their torque production during the entire task, and were instructed to focus on keeping their torque above zero. Subjects

were also specifically instructed to push as hard as they could during the last 15 seconds and not give up during the protocol, so that they did not begin to rest early. Subjects were given verbal encouragement and feedback on time remaining during the test.

During the validation protocol only, subjects then remained attached to the dynamometer and were asked to produce a set of three maximal isometric efforts into abduction in a neutral hip position every two minutes, until ten minutes total had elapsed after the fatigue protocol. This was performed to track the recovery of hip abduction strength. In addition, subjects self-reported their hip abductor fatigue on a visual analog scale (VAS) before the protocol, immediately after the protocol, and 10 minutes after the protocol by making a mark on a line 10 cm long, with 0 indicating no perceived fatigue in the hip abductors, and 10 indicating the most fatigue imaginable. VAS scores were then calculated on a scale of 1-10 for analysis.

Protocol – Running Study

To be eligible for the main running study, subjects needed to be between 18-45 years old (81,101), average running at least 20 miles per week (129), and report no major injuries for at least the previous 6 months (6,21). A major injury was consistent with the consensus definition by Yamato et al. (127). All subjects signed an informed consent form approved by the University of Oregon (Appendix B).

Subjects warmed up at their easy pace on a treadmill for five minutes. Hip abduction strength for the dominant limb was then tested in sidelying position on the Biodex System 3 dynamometer (Biodex Medical Systems, Shirley NY) (Figure 1). The dominant limb was again defined as the leg subjects would prefer to kick a soccer ball for distance. In total, hip abduction strength was tested four times throughout running study

protocol. Subjects were given real-time visual feedback of their hip abduction torque via a computer monitor for all tests.

Subjects were then outfitted with a vest attached to a 6-channel EMG backpack with adjustable gain (MA-300, Motion Lab Systems, Baton Rouge LA) (Figure 4A). Prior to electrode placement, the skin was lightly abraded and cleaned with an alcohol pad over the site of electrode placement locations. Six pre-amplified electrodes (Figure 4B) and one ground electrode were then placed directly on the skin. Conducting gel was placed between the skin and electrodes and several strips of white athletic tape secured the electrodes to the skin. The active electrodes were placed on the tensor fascia latae (TFL), gluteus medius (GMED) and gluteus maximus (GMAX) bilaterally. The positioning of electrodes can be seen in Figure 4C.

The EMG pre-amplifier contained a band-pass filter from 20 to 500 Hz and a gain of 20. The gain was then manually adjusted at the backpack, set at 4000 unless this produced signal clipping during the MVIC. This signal was then fed to a desktop amplifier (gain = 2) via a coaxial connection cable which transmitted the analog signal through a 12-bit A/D board connected to the motion capture computer in the Motion Analysis Laboratory. This signal was collected at 1000 Hz using Cortex 5 motion capture software (Motion Analysis Corporation, Santa Rosa CA) so that EMG and motion capture data would be synchronized. The signal to noise ratio was visually inspected as the subject performed standing hip abduction and hip extension. If the signal to noise ratio was not acceptable, slight adjustments were made to the electrode placement and the signal to noise ratio was retested.

Maximum voluntary isometric contractions (MVICs) were then performed on a padded treatment table. For the gluteus maximus MVIC, the subject was positioned prone with the knee flexed to 90° (116,117). Two stabilization straps were tightly attached just distal to the gluteal fold and just proximal to the popliteal fossa on one leg. A researcher also helped stabilize the subject by placing their hands on the thigh during the test. Subjects were instructed to push up against the straps using their gluteal muscle with maximal force for five seconds. This was repeated for the same leg after 45 seconds and was then repeated on the opposite leg.

MVICs for gluteus medius and TFL were performed with the subject sidelying and the hip in 0° of flexion and rotation, and 10° of abduction. Two stabilization straps were attached just proximal to the knee and just proximal to the ankle with tautness that would allow 10° of abduction. Similar to GMAX MVIC testing, a researcher helped stabilize their leg during testing to avoid the subject rotating their body. Subjects were instructed to push up against the straps using their hip abductor muscles with maximal force for five seconds. This was repeated for the same leg after 45 seconds and was then repeated on the opposite leg.

Subjects were then outfitted with 39 reflective markers (6,41). The trunk segment was defined by two markers on the acromion processes, as well as a virtual marker at the pelvis center of mass (81). The pelvis was defined by two markers on the anterior superior iliac spines, and one at the midpoint between the posterior superior iliac spines. The femur was defined by two markers at the medial and lateral femoral epicondyles, and one marker mid-thigh in line with the lateral epicondyle and greater trochanter. The hip joint center was defined by measurements of ASIS breadth (112). The shank was defined

by the epicondyle markers, two markers on the medial and lateral malleoli, and a marker on the medial shank.

Subjects then ran continuous laps of approximately 40-meters in the Motion Analysis Laboratory at their easy run pace while whole body kinematics were collected with a 10-camera motion capture system (Motion Analysis Corp., Santa Rosa CA) sampling at 200 Hz. This pace was defined as the pace a subject could comfortably maintain for 30 minutes while maintaining a conversation. Participants were instructed not to alter their stride to hit three force plates (AMTI, Watertown, MA) located in series in this region. Subjects were given two laps to become accustomed to running in the lab before data was collected. Subjects then completed 20 laps in the laboratory. Data were collected when the participants passed through a straight 10-meter region in the center of the capture volume.

After running 20 laps in the lab, hip abduction strength was tested again on the dynamometer. For this test, subjects only pushed one time for five seconds. If subjects did not meet or exceed their previous maximum hip abduction torque, the test was performed one more time.

Subjects were then given instructions for the fatigue protocol. The instructions and procedures for the protocol itself were the exact same as during the protocol validation. Immediately after the two-minute fatigue protocol, within approximately 3-5 seconds, subjects performed one maximum isometric effort in the direction of abduction. This was done to assess the decrease in hip abduction torque immediately following the protocol.

Subjects then immediately were detached from the Biodex machine and began running again in the lab. Twenty laps were again completed while whole body kinematics were collected by the motion capture system. After running, subjects performed one more isometric hip abduction strength test – three efforts separated by fifteen seconds.

Data Processing and Analysis

To quantify hip abduction strength at the first and last time point (three efforts), mean maximal torque was calculated by averaging the peak torque generated during each three effort. To quantify hip abduction strength during the second and third time points, the maximum torque generated during the one five-second effort was calculated. For all tests, the hip abduction torque was normalized by body mass for analysis.

Five running trials per limb per subject were selected for analysis (116,117) for both pre-fatigue and post-fatigue running. Only trials where the foot cleanly struck a force plate were selected. Foot strike was defined as the first frame where the vertical ground reaction force exceeded 50 Newtons (N), and toe off was defined as the first frame where the vertical ground reaction force fell below 50 N (14). During the post-fatigue running task, all trials analyzed were within the first 10 laps to minimize any kinematic changes as hip abduction strength recovered while running.

Marker trajectories were identified using Cortex 5.0 motion capture software (Motion Analysis Corp., Santa Rosa CA) and were smoothed using a low-pass, fourth-order, zero-lag Butterworth filter with an 8 Hz cutoff. A custom LabView program (National Instruments, Austin TX) calculated hip angles during stance phase using a YXZ cardan angle rotation sequence. Angular excursions and peak angles in all the

planes at the knee, hip, pelvis, and trunk were calculated. Peak angles were defined as the maximum joint or segment angle during stance phase. Angular excursion was defined as the difference between the angle at initial contact and the peak angle during stance phase (4). Excursions and peak angles were calculated for each trial and averaged for analysis.

All EMG data were processed and analyzed using a custom LabView program. EMG data were band-pass filtered from 20-450 Hz, root-mean-square (RMS) averaged and smoothed using a 50 ms sliding window. To determine the MVIC for each muscle, both trials were processed. For both MVIC attempts, a sliding 500 ms window calculated the mean amplitude of the highest signal portion of the MVIC trial. The highest value between trials was determined the MVIC value for each muscle.

Running trials were trimmed to 250 ms before foot strike and 250 ms after toe off, respectively, for analysis. Due to variability in the data, no set threshold above baseline was determined feasible to determine EMG onset. Therefore, EMG onset and offset was defined by visual inspection, which has shown to be reliable (53). EMG onset was defined as the first major vertical deflection of the EMG signal (53) during the 250 ms window before heel strike which remained above baseline for at least 25 ms (116,117). EMG offset was defined as the last major vertical deflection of the EMG signal (53) which remained below baseline for at least 25 ms (116,117). EMG onset is represented numerically as the time in milliseconds before foot strike, while EMG offset is represented numerically as the time in milliseconds before toe-off (Figure 5). EMG duration was calculated as the difference between the onset and offset times (Figure 5)

EMG peak amplitude was calculated as the maximum EMG recording within the visually determined onset and offset times (Figure 5). This maximum EMG value was normalized by the MVIC for that muscle. All four EMG parameters were then averaged between the five trials for analysis. In rare cases where the mean peak amplitude exceeded 200% MVIC, this data point was discarded for analysis, as it was assumed the subject was not able to perform a true MVIC for that muscle.

Statistical Analysis

A 2 x 2 mixed effects ANCOVA was calculated for peak joint angles, joint angle excursions, and EMG parameters to determine the effect of the fatigue protocol and sex on the dependent variables. Because of a significant difference in running speed between bouts of running, percent change in running speed was entered as a covariate in the analysis. An alpha-level of .05 was set for all omnibus tests. Because each factor only contained two levels, no Bonferroni corrections for pairwise comparisons were warranted. All statistics were calculated using SPSS version 23 (SPSS Inc., Chicago IL), except for effect sizes, which were calculated using G*Power 3.1 (G*Power, Düsseldorf, Germany).

Results

Fatigue Protocol Validation

Twelve subjects met the inclusion criteria and agreed to participate in the study fatigue protocol validation study, six males and six females (age: 25.5 ± 4.7 years, height: 175.6 ± 6.8 cm, weight: 70.5 ± 9.8 kg). On average, hip abduction torque immediately after the fatigue protocol significantly decreased by $27.4 \pm 9.9\%$, $p < .001$. Over the

course of 10 minutes, torque recovered fairly linearly, with hip abduction torque still $10.6 \pm 6.6\%$ decreased compared to baseline, $p = .001$ (Figure 6).

VAS scores significantly increased from before the fatigue protocol (0.43 ± 0.55) to immediately after the fatigue protocol (7.44 ± 1.99), $p < .001$. VAS scores remained elevated 10 minutes after the protocol (5.43 ± 2.79), which was still significantly greater than before the fatigue protocol, $p < .001$ (Figure 7).

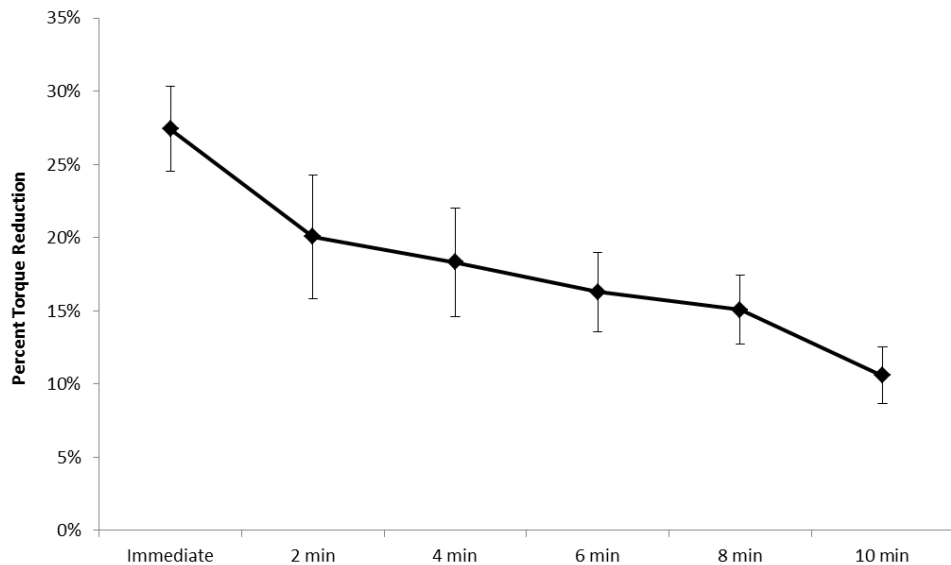


Figure 6. Average percent reduction in hip abduction torque immediately following the two minute fatigue protocol, and every two minutes for ten minutes.

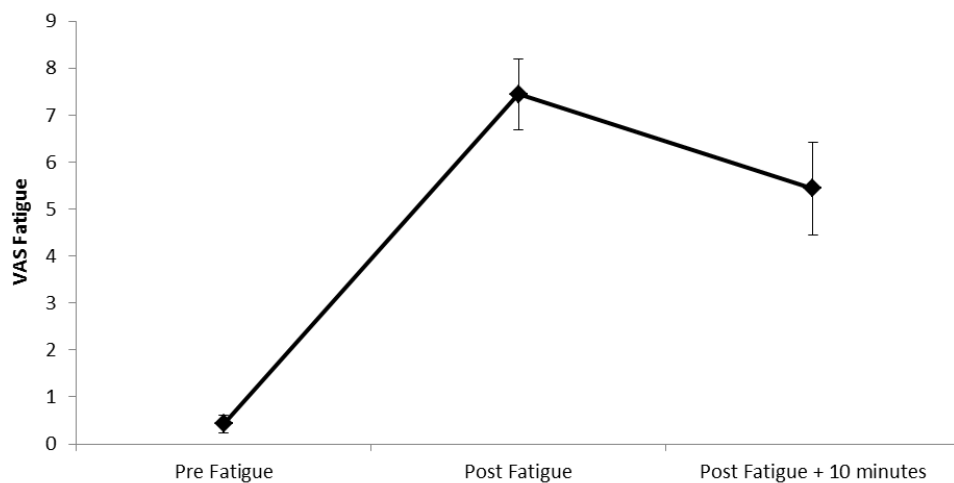


Figure 7. Visual analog scale perception of fatigue immediately before, immediately after, and ten minutes after the fatigue protocol.

Running Study

Thirty subjects (15 males and 15 females) met the inclusion criteria and were included in this study. Subject demographics can be seen in Chapter II (Table 1).

Hip abduction torque significantly decreased by an average of $31.8 \pm 15.5\%$ following the fatigue protocol ($p < .001$, effect size = 1.56), and remained significantly lower ($9.1 \pm 9.4\%$) after the second bout of running ($p < .001$, effect size = 0.80) (Figure 8). There was no interaction effect between time and sex, nor main effect of sex. On average, there was 44.0 ± 12.9 seconds between the end of the fatigue protocol and when subjects began the second bout of running, and 6 minutes and 52 seconds (± 96 seconds) between the end of the fatigue protocol and the final hip abduction strength test. Running speed did significantly increase from before the fatigue protocol (2.98 ± 0.28 m/s) to after the fatigue protocol (3.07 ± 0.29 m/s), $p = .004$.

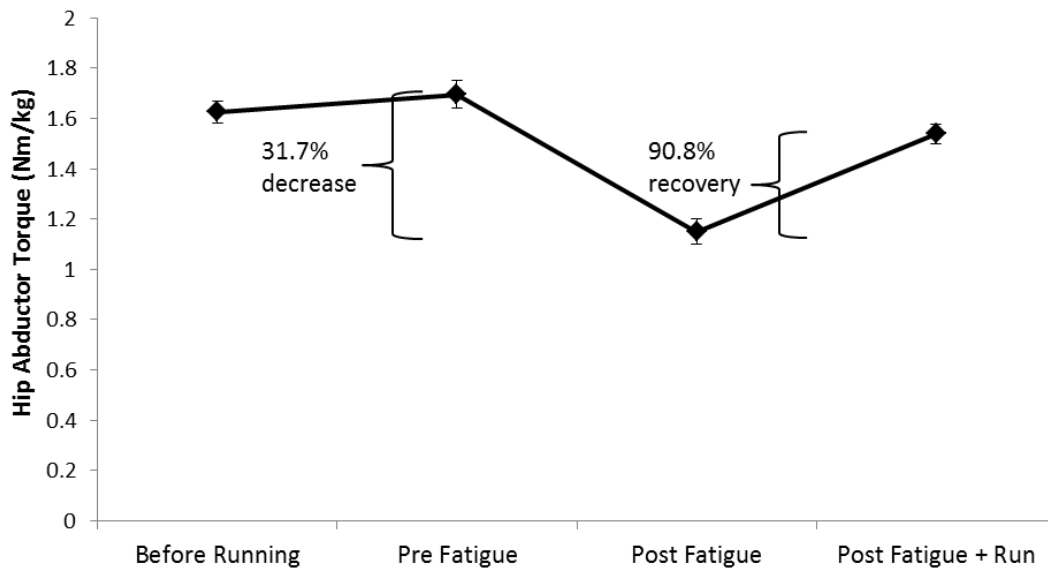


Figure 8. Hip abduction strength measured at four distinct time points

There was no significant interaction effect for peak angles at the knee, hip, or pelvis. There was a significant fatigue by sex interaction for trunk flexion ($p = .004$), as trunk flexion significantly decreased following fatigue in males (pre-fatigue: 9.61° ; post-fatigue: 8.80° ; $p = .035$), and increased following fatigue in females (pre-fatigue: 8.90° ; post-fatigue: 9.84° ; $p = .021$). Peak knee flexion significantly decreased ($p = .002$), while peak ipsilateral trunk lean significantly increased ($p = .006$) after the fatigue protocol (Figure 9; Table 12). There was no main effect of sex for any variable.

There were no interaction effects for any joint excursions at the knee, hip, pelvis, or trunk. Pelvic rotation excursion ($p = .011$) and ipsilateral trunk lean excursion ($p = .011$) significantly increased after the fatigue protocol, while trunk rotation excursion significantly decreased ($p = .008$) (Figure 10; Table 13). In addition, females demonstrated greater hip rotation excursion (females: $8.45 \pm 3.38^\circ$; males: $6.00 \pm 2.89^\circ$; $p = .040$), trunk flexion excursion (females: $3.95 \pm 1.34^\circ$; males: $2.90 \pm 1.16^\circ$; $p = .031$), and trunk rotation excursion (females: $27.87 \pm 5.35^\circ$; males: $21.15 \pm 4.35^\circ$ $p = .002$) compared to males.

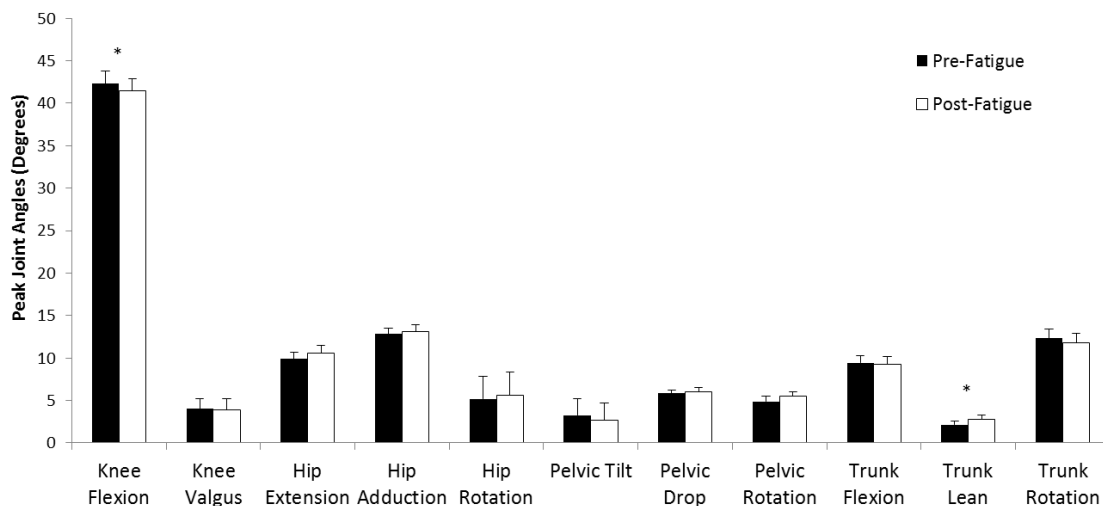


Figure 9. Peak angles of the knee, hip, pelvis, and trunk before and after the hip abductor fatigue protocol

Table 12. Peak joint angles before and after the hip abductor fatigue protocol

Peak Angles (°)	Pre-Fatigue	Post-Fatigue	<i>p</i> -value	Effect Size
Knee Flexion	42.29 ± 5.86	41.51 ± 5.49	0.002*	0.654
Knee Valgus	4.00 ± 6.03	3.92 ± 6.18	0.847	0.061
Hip Extension	9.90 ± 3.55	10.53 ± 4.98	0.681	0.294
Hip Adduction	12.83 ± 3.04	13.06 ± 3.48	0.812	0.154
Hip Internal Rotation	5.20 ± 10.6	5.65 ± 10.8	0.381	0.144
Pelvic Tilt	3.27 ± 6.53	2.72 ± 6.93	0.151	0.231
Pelvic Drop	5.84 ± 2.17	5.98 ± 2.71	0.569	0.103
Pelvic Rotation	4.86 ± 3.33	5.50 ± 3.41	0.955	0.305
Trunk Flexion	9.42 ± 3.38	9.30 ± 3.52	0.471	0.019
Trunk Lean	2.14 ± 1.73	2.83 ± 1.87	0.006*	0.773
Trunk Rotation	12.35 ± 4.12	11.78 ± 3.86	0.140	0.092

* Indicates a significant difference between conditions, $p < .05$.

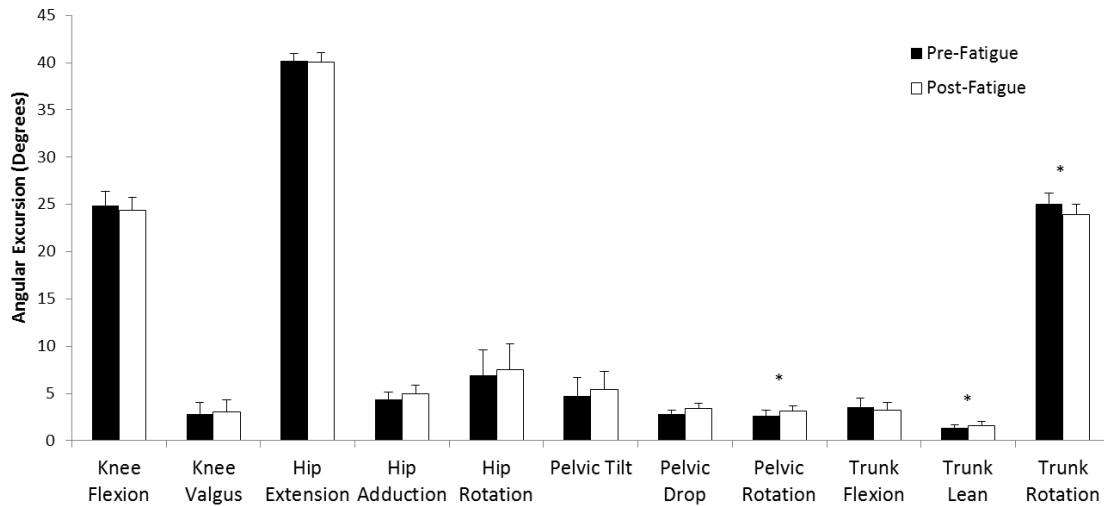
**Figure 10.** Angular excursions of the knee, hip, pelvis, and trunk before and after the hip abductor fatigue protocol

Table 13. Angular excursions before and after the hip abductor fatigue protocol

Excursions (°)	Pre-Fatigue	Post-Fatigue	<i>p</i>-value	Effect Size
Knee Flexion	24.84 ± 4.34	24.33 ± 4.17	0.099	0.330
Knee Valgus	2.79 ± 3.28	3.01 ± 3.24	0.225	0.209
Hip Extension	40.17 ± 3.81	40.08 ± 4.46	0.260	0.038
Hip Adduction	4.41 ± 2.36	4.95 ± 2.75	0.283	0.278
Hip Internal Rotation	6.90 ± 3.37	7.55 ± 3.53	0.720	0.239
Pelvic Tilt	4.74 ± 1.43	5.42 ± 1.79	0.163	0.466
Pelvic Drop	2.84 ± 1.33	3.46 ± 1.89	0.244	0.359
Pelvic Rotation	2.60 ± 2.70	3.14 ± 2.81	0.011*	0.686
Trunk Flexion	3.56 ± 1.21	3.23 ± 1.33	0.108	0.516
Trunk Lean	1.32 ± 0.77	1.58 ± 0.84	0.018*	0.681
Trunk Rotation	24.89 ± 5.35	23.89 ± 5.83	0.008*	0.414

* Indicates a significant difference between sexes, $p < .05$

There were no interaction effects for any EMG variable. TFL offset significantly decreased (i.e., occurred closer to toe off during stance phase) ($p = .022$) and TFL duration significantly increased ($p = .045$) after the fatigue protocol. GMAX peak amplitude ($p = .050$) and GMAX duration ($p = .032$) also significantly increased after the fatigue protocol (Table 14). Finally, GMAX onset occurred significantly earlier in males compared to females (females: 30.38 ± 24.90 ms; males: 61.10 ± 26.6 ms; $p = .004$).

Because we did not see any differences in peak angles or excursions for hip adduction and hip internal rotation, but did see changes at the knee, pelvis, and trunk, we performed post-hoc correlations between the percent decrease in hip abduction torque and percent increase for several kinematic variables (Tables 15-16). There was a significant correlation between the percent decrease in hip abduction torque, and the percent increase in peak trunk lean ($r = 0.394$, $p = .030$) (Table 16).

Table 14. EMG variables before and after the hip abductor fatigue protocol

	Pre-Fatigue	Post-Fatigue	<i>p</i> -value	Effect Size
TFL Amplitude	94.37 ± 55.11	104.68 ± 81.71	0.462	0.198
TFL Onset	40.83 ± 33.05	39.21 ± 26.29	0.762	0.101
TFL Offset	78.41 ± 21.23	64.71 ± 24.75	0.022*	0.628
TFL Duration	209.72 ± 39.30	216.93 ± 35.83	0.045*	0.270
GMED Amplitude	86.31 ± 44.42	86.73 ± 38.84	0.645	0.008
GMED Onset	21.90 ± 25.15	24.93 ± 35.56	0.339	0.094
GMED Offset	78.90 ± 26.75	72.68 ± 31.19	0.809	0.295
GMED Duration	192.03 ± 28.98	195.14 ± 39.00	0.427	0.083
GMAX Amplitude	75.75 ± 34.20	90.72 ± 49.58	0.050*	0.389
GMAX Onset	42.43 ± 28.46	48.83 ± 30.84	0.107	0.302
GMAX Offset	70.83 ± 20.37	61.03 ± 29.38	0.086	0.394
GMAX Duration	218.80 ± 34.15	230.52 ± 34.18	0.032*	0.347

(Amplitude = % MVIC; Onset, Offset, Duration = ms)

* Indicates a significant difference between sexes, $p < .05$.

Table 15. Correlations between % decrease in hip abduction strength and % increase in hip kinematics before and after the hip abductor fatigue protocol

	Hip Adduction Excursion	Peak Hip Adduction	Hip Internal Rotation Excursion	Peak Hip Internal Rotation
% Decrease	$r = 0.072$	$r = 0.072$	$r = -0.206$	$r = 0.132$

Table 16. Correlations between % decrease in hip abduction strength and % increase in pelvis and trunk kinematics before and after the hip abductor fatigue protocol

	Pelvic Rotation Excursion	Trunk Lean Excursion	Peak Trunk Lean	Peak Knee Flexion
% Decrease	$r = 0.021$	$r = 0.333$	$r = 0.394^*$	$r = 0.132$

* Indicates a significant correlation, $p < .05$.

Discussion

The main purpose of this study was to investigate changes in running kinematics and hip muscle activation after a hip abductor fatigue protocol. The hip abductor fatigue protocol significantly decreased hip abduction torque immediately after the protocol, and remained significantly lower after the second bout of running. However, there were no significant changes in the amplitude or timing of the gluteus medius, the primary hip abductor muscle, and no significant differences in kinematics at the hip. Small changes at the knee, pelvis, and trunk, as well as alterations in TFL and GMAX muscle activity, were seen following the fatigue protocol.

Because the fatigue protocol consisted of repeated, maximal eccentric contractions for the hip abductors, this protocol likely induced muscular damage, especially in the gluteus medius. In addition, metabolites likely accumulated in the hip abductor muscles during the protocol. The decreased capacity of the hip abductor muscles to produce force after the protocol is probably due to a combination of these factors. Despite the probability of muscle damage and metabolite buildup, gluteus medius peak amplitude and timing during running gait were not affected. Other kinematic and EMG variables, however, appear to have been affected by the decrease in hip abduction strength.

Both peak ipsilateral trunk lean and trunk lean excursion increased following the fatigue protocol, with moderate effect sizes for both variables (Figure 9; Table 13). While the absolute changes in trunk lean magnitude were relatively small, peak trunk lean increased in 25 out of 30 subjects, and the correlation between the percent decrease in hip abduction strength and the percent increase in peak trunk lean after the fatigue

protocol was significant (Table 16), indicating that hip abduction strength may be a significant modulator of frontal plane trunk motion during running.

Increased lateral bending of the trunk towards the stance limb should decrease the moment arm of the center of mass relative to the hip joint center (91). Thus, the increase in trunk lean towards the stance limb after hip abductor strength is decreased is likely an attempt to offload the hip abductor muscles during running. Due to the influence of frontal plane trunk position on the COM moment arm, these small alterations in trunk position could have a substantial impact on frontal plane hip and knee moments and impulses during running (103,105). While this study did not look at knee joint moments, previous work indicates that isolated hip abductor fatigue does shift the external knee joint moment in the valgus direction (37). This biomechanical compensation is theorized to increase the tensile stress on the knee, possibly increasing the risk of significant injury (91).

Peak knee flexion significantly decreased following the hip abductor fatigue protocol, meaning the knee was slightly more extended during midstance. Lenhart et al. (2014) reported a significant positive correlation with peak knee flexion angle and patellofemoral joint force (64). Thus, the decreased peak knee flexion angle observed in this study would likely mean that peak patellofemoral force at the knee decreased following the fatigue protocol. If greater ipsilateral trunk lean has negative consequences to knee joint forces, decreasing knee flexion could be an attempt to counteract the deleterious changes at the trunk.

We observed a fatigue by sex interaction effect for peak trunk flexion, as peak trunk flexion decreased in males and increased in females after the fatigue protocol.

GMAX peak amplitude also increased in both males and females after the fatigue protocol, and GMAX onset was overall earlier in males than females. In addition to being the primary hip extensor muscle during running, gluteus maximus also acts to control forward trunk flexion during running (66). Gluteus maximus amplitude has a strong negative correlation with the rate of trunk flexion during the first half of stance phase (66), meaning that greater amplitude is associated with slower rates of flexion. Because of the decrease in hip abduction strength, an increase in GMAX peak amplitude may be a compensatory mechanism to increase trunk stability during running. In males, it thus makes sense that GMAX peak amplitude increased while peak trunk flexion decreased after fatigue, especially given that GMAX onset occurred earlier overall in males.

The relationship between GMAX amplitude and peak trunk flexion following the fatigue protocol in females, however, appears somewhat paradoxical. Despite an increase in GMAX peak amplitude, females also demonstrated increased peak trunk flexion after the fatigue protocol. Females, on average, ran with greater trunk flexion than males (35,46,97), and also may run with greater peak GMAX amplitude relative to their MVIC compared to males (116). One possible explanation is that in females, gluteus maximus is recruited at a higher percent of maximum, partially as a result of trying to control excessive trunk flexion, which may have been exacerbated after the fatigue protocol. It is also possible that recruiting gluteus maximus at a higher percentage of maximum during running means that gluteus maximus fatigue occurs sooner, resulting in greater trunk flexion. Greater amounts of trunk flexion are not bad however – quite the opposite, as increasing trunk flexion is associated with decreasing patellofemoral joint stress, and thus

may be injury-protective (108). It is interesting that females, who have a higher risk of developing patellofemoral pain, demonstrate an injury-protective movement toward greater peak trunk flexion. The relationship between trunk flexion during running, GMAX amplitude during running, and the risk of knee injury in females appears to require further research.

Pelvic rotation excursion increased after the fatigue protocol in both sexes, which is not surprising given gluteus medius is thought to help control transverse plane pelvis motion during locomotion (39). Hip abduction strength has also been moderately correlated with pelvic rotation excursion in males during running (46). Thus, decreasing hip abduction strength appears to limit the ability of the hip abductor muscles to control pelvis motion in the transverse plane.

Trunk rotation excursion significantly decreased in both sexes after the fatigue protocol, and trunk rotation excursion was greater in females compared to males overall. Thus, trunk rotation in all three planes appears to be affected by decreasing hip abduction strength. Peak trunk rotation generally occurs at toe off. Decreased trunk rotation excursion, but not peak trunk rotation, thus may indicate that the trunk is slightly more rotated toward the stance limb at foot strike, which may also be an attempt to offload the hip abductor muscles.

TFL duration increased, and TFL offset time occurred significantly closer to toe-off. This compensation may be an attempt of the other major hip abductor muscle to provide stability to the hip joint after maximum strength was compromised.

Given these changes in kinematics and EMG, it is still unclear why GMED parameters did not change. Greater trunk lean should decrease the amount of force

gluteus medius needed to contract. While force generally scales with EMG (either linearly or non-linearly), this relationship is not as clear under dynamic activities, and at higher percentages of MVIC (58). It is possible that the combination of muscle damage and neuromuscular fatigue was not high enough to affect the nervous input to the muscle. It is also possible that the subjects, knowing their muscle strength was diminished, attempted to consciously recruit their affected hip abductor muscles, overcoming any deficits in nervous input after fatigue.

It is also important to point out that while hip kinematics did not change overall, some subjects did have considerable changes to hip kinematics. Every subject likely compensates differently to the hip abductor fatigue protocol, depending on many factors, not limited to their relative strength pre-fatigue and the amount of torque reduction after fatigue. Further analyses could look to identify subgroups of runners based on their compensation patterns.

This study is not without several limitations. First, running speed before and after fatigue was not tightly controlled. While runners were instructed to run at the same pace, several runners slightly increased their running speed after the fatigue protocol, likely due to adrenaline. Running speed was accounted for as a covariate in the statistical analysis to help mitigate this difference. In addition, there was almost a significant difference in average mileage between males and females, with males averaging almost 10 miles more per week compared to females. Thus, we cannot say for certain whether some sex differences may be partly due to relative training volume. Foot strike patterns were not controlled in this study, so we cannot say whether any changes after hip abductor fatigue vary per foot strike pattern. In addition, all subjects wore their self-selected shoes, so that

introducing a new shoe was not a confounding variable. It is unclear whether a different shoe type could mitigate or enhance any effects of decreased hip abduction strength. Finally, surface EMG measures have several inherent limitations, such as muscle cross talk, movement artifact, and electrode placement errors, which can negatively affect the quality of EMG data.

In conclusion, decreasing hip abduction strength in runners did not change gluteus medius amplitude or timing, and did not affect hip joint kinematics. However, we did see changes in both distal and proximal kinematics, and gluteus maximus and TFL EMG, which may have injury implications.

Bridge

Chapters II-IV have investigated factors related to the neuromuscular control of the hip, pelvis, and trunk from a traditional biomechanical and neurophysiological perspective. Chapter V uses a different technique to probe the neuromuscular control of the hip, called continuous relative phase, which can quantify coordination between adjacent segments. This technique can be seen as an alternative method to investigate neuromuscular control of the hip.

CHAPTER V

DOES HIP ABDUCTION STRENGTH OR SEX AFFECT COORDINATIVE VARIABILITY AT THE HIP DURING RUNNING?

This chapter contains co-authored material. JJ Hannigan was responsible for the conceptual development, development of the protocol, data collection and analysis, and writing of the manuscript. Dr. Li-Shan Chou contributed to the conceptual development and refinement of the protocol, and provided critiques and editing for the manuscript.

Introduction

Traditional biomechanical analyses of human movement generally involve quantifying kinematic or kinetic parameters, such as individual joint angles, velocities, or moments. While this approach has successfully identified risk factors for overuse injury, the underlying mechanisms for injury and relationship between injury factors has remained elusive, likely because most injuries are multifactorial (44). To combat this issue, some biomechanists over the past 20 years have begun studying injuries from a dynamical systems approach, which investigates human movement on a more macroscopic level, taking into account the contribution of multiple injury factors (43).

While there are several techniques for quantifying coordination in the dynamical system approach, such as vector coding and discrete relative phase, continuous relative phase (CRP) has recently gained support in the biomechanics community, specifically in the field of overuse and running-related injuries. One reason for its preference is the inclusion of both temporal and spatial information in its calculation, which provides “higher-dimensional” information compared to other measures (42,44). Second, CRP

can take into account the entire gait cycle or stance phase of any movement, unlike other measures, which only analyze coupling at discrete time points during a movement (42,44).

Central to the dynamical systems approach is the concept of coordinative variability, which needs to be distinguished from end-point variability. Some tasks require low end-point variability, where the end result of a task must be tightly controlled, such as hitting a bulls-eye in darts. In contrast, coordinative variability refers to the coordination of all joints and segments required to produce movement to perform the task (42). Continuing with the darts example, this would refer to the coordinated movement between the shoulder, elbow, wrist, hand, and fingers needed to throw the dart. While experts at a task such as dart-throwing may display lower end-point variability than novices, evidence suggests that experts may display greater coordinative variability than novices for that task (2).

Low coordinative variability also appears related to injury risk. In theory, low coordinative variability between adjacent segments may lead to repetitive loading of the same structures and has been implicated in several overuse injuries (42,44,69). In runners with PFPS, decreased coordinative variability was found between the thigh and shank and between the shank and foot segment (18,42). In addition, females have demonstrated decreased coordinative variability compared to males during an unanticipated cutting maneuver, which may help explain the female sex bias for ACL tears (89).

Despite the proposed link between both hip abduction strength (11,23,52,71,93,101) and hip kinematics (74,80–82,101,115,119) on PFPS injury risk, as

well as the female sex bias for PFPS (106), no study to date has looked at CRP variability at the hip, analyzed the relationship between hip strength and CRP variability, or compared CRP variability between sex during running. Therefore, the primary purposes of this study were to a) investigate the effect of decreasing hip abduction strength on CRP variability during running, and b) compare CRP variability at the hip between sexes. It was hypothesized that CRP variability would increase after hip abductor fatigue, and be lower in females compared to males.

Methods

To be included in the study, subjects needed to be between 18-45 years old (81,101), average running at least 20 miles per week (129), and report no major injuries for at least the previous 6 months (6,21). A major injury was consistent with the consensus definition by Yamato et al. (127). All subjects signed an informed consent form approved by the University of Oregon (Appendix B).

Subjects warmed up at their easy pace on a treadmill for five minutes. Hip abduction strength for the dominant limb was then tested in sidelying position on the Biodex System 3 dynamometer (Biodex Medical Systems, Shirley NY) (Figure 1). The dominant limb was again defined as the leg subjects would prefer to kick a soccer ball for distance. In total, hip abduction strength was tested four times throughout running study protocol. Subjects were given real-time visual feedback of their hip abduction torque via a computer monitor for all tests.

Subjects were then outfitted with 39 reflective markers (6,41). The pelvis was defined by two markers on the anterior superior iliac spines, and one at the midpoint

between the posterior superior iliac spines. The femur was defined by two markers at the medial and lateral femoral epicondyles, and one marker mid-thigh in line with the lateral epicondyle and greater trochanter. The hip joint center was defined by measurements of ASIS breadth (112).

Subjects then ran continuous laps of approximately 40-meters in the Motion Analysis Laboratory at their easy run pace while whole body kinematics were collected with a 10-camera motion capture system (Motion Analysis Corp., Santa Rosa CA) sampling at 200 Hz. This pace was defined as the pace a subject could comfortably maintain for 30 minutes while maintaining a conversation. Participants were instructed not to alter their stride to hit three force plates (AMTI, Watertown, MA) located in series in this region. Subjects were given two laps to become accustomed to running in the lab before data was collected. Subjects then completed 20 laps in the laboratory. Data were collected when the participants passed through a straight 10-meter region in the center of the capture volume.

After running 20 laps in the lab, hip abduction strength was tested again on the dynamometer. For this test, subjects only pushed one time for five seconds. If subjects did not meet or exceed their previous maximum hip abduction torque, the test was performed one more time.

Subjects were then positioned sidelying on the padded treatment table with the greater trochanter of the dominant leg aligned with the dynamometer arm and the resistance pad attached firmly to the thigh three finger lengths above the joint line. Subjects were then given explicit instructions for the fatigue protocol, and researchers ensured subjects were comfortable with the protocol before proceeding.

The fatigue protocol was a dynamic two-minute task, as the arm of the dynamometer rotated at 30° per second through a 30° range of motion, regardless of the subject's exerted torque. All subjects were instructed to push in the direction of abduction for two minutes while the dynamometer rotated through this range of motion, with specific instructions to try to resist the dynamometer as it rotated downward. Thus, when the dynamometer rotated upward, the hip abductor muscles were contracting concentrically, and when the dynamometer rotated downward, the hip abductor muscles were contracting eccentrically. Subjects were given visual feedback of their torque production during the entire task, and were instructed to focus on keeping their torque above zero. Subjects were also specifically instructed to push as hard as they could during the last 15 seconds and not give up during the protocol, so that they did not begin to rest early. Subjects were given verbal encouragement and feedback on time remaining during the test.

Immediately after the two-minute fatigue protocol, within approximately 3-5 seconds, subjects performed one maximum isometric effort in the direction of abduction. This was done to assess the decrease in hip abduction torque immediately following the protocol. Subjects then immediately were detached from the Biodex machine and began running again in the lab. Twenty laps were again completed while whole body kinematics were collected by the motion capture system. After running, subjects performed one more isometric hip abduction strength test – three efforts separated by fifteen seconds.

Data Processing and Analysis

To quantify hip abduction strength at the first and last time point (three efforts), mean maximal torque was calculated by averaging the peak torque generated during each three efforts. To quantify hip abduction strength during the second and third time points, the maximum torque generated during the one five-second effort was calculated. For all tests, the hip abduction torque was normalized by body mass for analysis.

Five running trials per limb per subject were selected for analysis (116,117) for both pre-fatigue and post-fatigue running. Only trials where the foot cleanly struck a force plate were selected. Foot strike was defined as the first frame where the vertical ground reaction force exceeded 50 Newtons (N), and toe off was defined as the first frame where the vertical ground reaction force fell below 50 N (14). During the post-fatigue running task, all trials analyzed were within the first 10 laps to minimize any kinematic changes as hip abduction strength recovered while running.

Marker trajectories were identified using Cortex 5.0 motion capture software (Motion Analysis Corp., Santa Rosa CA) and were smoothed using a low-pass, fourth-order, zero-lag Butterworth filter with an 8 Hz cutoff. A custom LabView program (National Instruments, Austin TX) calculated pelvis and thigh angles and velocities during stance phase with respect to a global coordinate system using a YXZ cardan angle rotation sequence. These values were interpolated to 100% of stance phase and normalized to values between -1 and 1 (13) using the following formulas (42):

$$\theta_i = \frac{2 * [\theta_i - \min(\theta_i)]}{\max(\theta_i) - \min(\theta_i)}$$

$$\omega_i = \frac{\omega_i}{\max\{\max(\omega_i), \max(-\omega_i)\}}$$

Phase portraits were constructed from these normalized segment angles (θ) (x-axis) and velocities (ω) (y-axis) so that phase angles (ϕ) could be calculated using the following formula:

$$\phi = \tan^{-1}(\omega/\theta)$$

Continuous relative phase (CRP) was then calculated by subtracting the phase angle of the thigh segment from the pelvis segment in both the frontal and transverse planes, respectively (42). All trials for each subject were averaged to create an ensemble CRP curve for all three planes of motion, both pre and post-fatigue protocol. Point-by-point CRP variability was calculated by averaging the standard deviation at each percent of stance phase (42). The overall variability of the CRP was also quantified for each runner by averaging the point-by-point variability for all 101 data points both before and after the fatigue protocol (15).

Statistical Analysis

A three-way mixed effects ANCOVA was used to calculate the effect of sex, time (each 10% of stance phase), and fatigue (pre-fatigue and post-fatigue) on CRP variability of the dominant limb. Because of a significant difference in running speed between bouts of running, percent change in running speed was entered as a covariate in the analysis. An alpha-level of .05 was set for all omnibus tests.

To more closely analyze the effect of sex, both limbs were analyzed before the fatigue protocol using a mixed effects ANOVA. The within-subjects factor was time (each 10% of stance phase), and the between subjects factors was sex. All statistics were calculated using SPSS version 23 (SPSS Inc., Chicago IL).

Results

Thirty subjects (15 males and 15 females) met the inclusion criteria and were included in this study. Subject demographics can be seen in Chapter II (Table 1). The fatigue protocol significantly decreased hip abduction strength (Chapter IV, Figure 8).

In the sagittal plane, there were no main interaction effects between any variable, and no main effect of fatigue or sex, $p > .05$ for all omnibus tests. There was a significant main effect of time, $p = .002$. Due to the number of pairwise comparisons, and relative unimportance of only comparing coordinative variability across stance phase for all subjects, these pairwise comparisons are not presented. Sagittal plane CRP variability during stance phase in males and females both before and after the fatigue protocol can be seen in Figure 11.

In the frontal plane, there was a significant time by fatigue interaction, $p = .037$. Follow-up pairwise comparisons revealed that CRP variability significantly increased from 20-40% of stance phase after the fatigue protocol. While the sex by time by fatigue interaction was not quite significant, pairwise comparisons in this analysis revealed that this increase in CRP variability from 20-40% of stance phase was mainly driven by increased variability in females. Also in support of this claim, there was also a trending, but non-significant fatigue by sex interaction, $p = .066$. Follow-up pairwise comparisons suggested that CRP variability increased in females after the fatigue protocol (pre-fatigue: $22.38 \pm 11.54^\circ$; post-fatigue: $29.92 \pm 11.85^\circ$) (Figure 12).

In the transverse plane, no significant interaction or main effects were seen for any variable. Transverse plane CRP variability during stance phase in males and females both before and after the fatigue protocol can be seen in Figure 13.

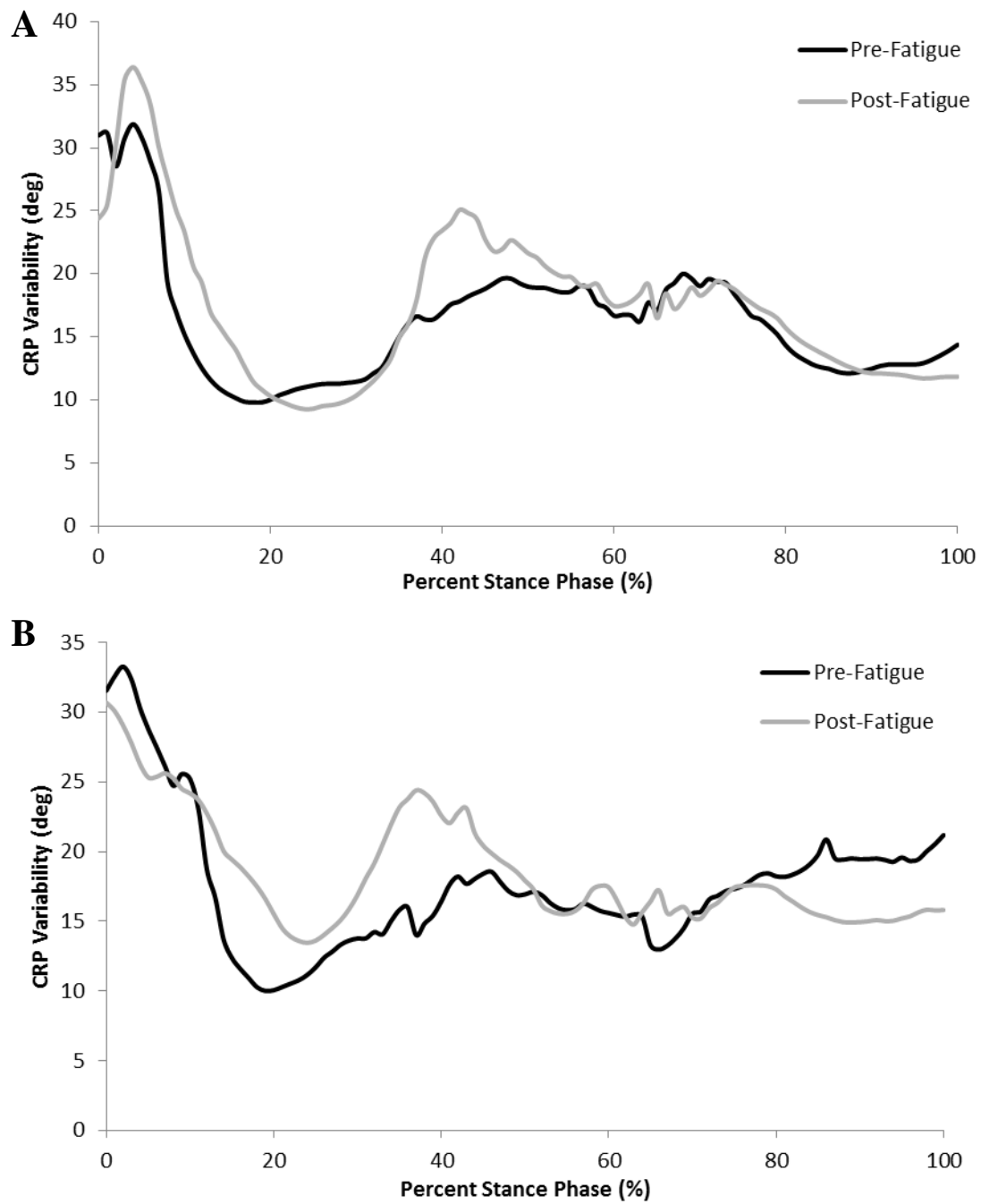


Figure 11. Sagittal plane CRP variability before and after fatigue in **A)** females and **B)** males. There were no interaction effects or main effects of sex or fatigue.

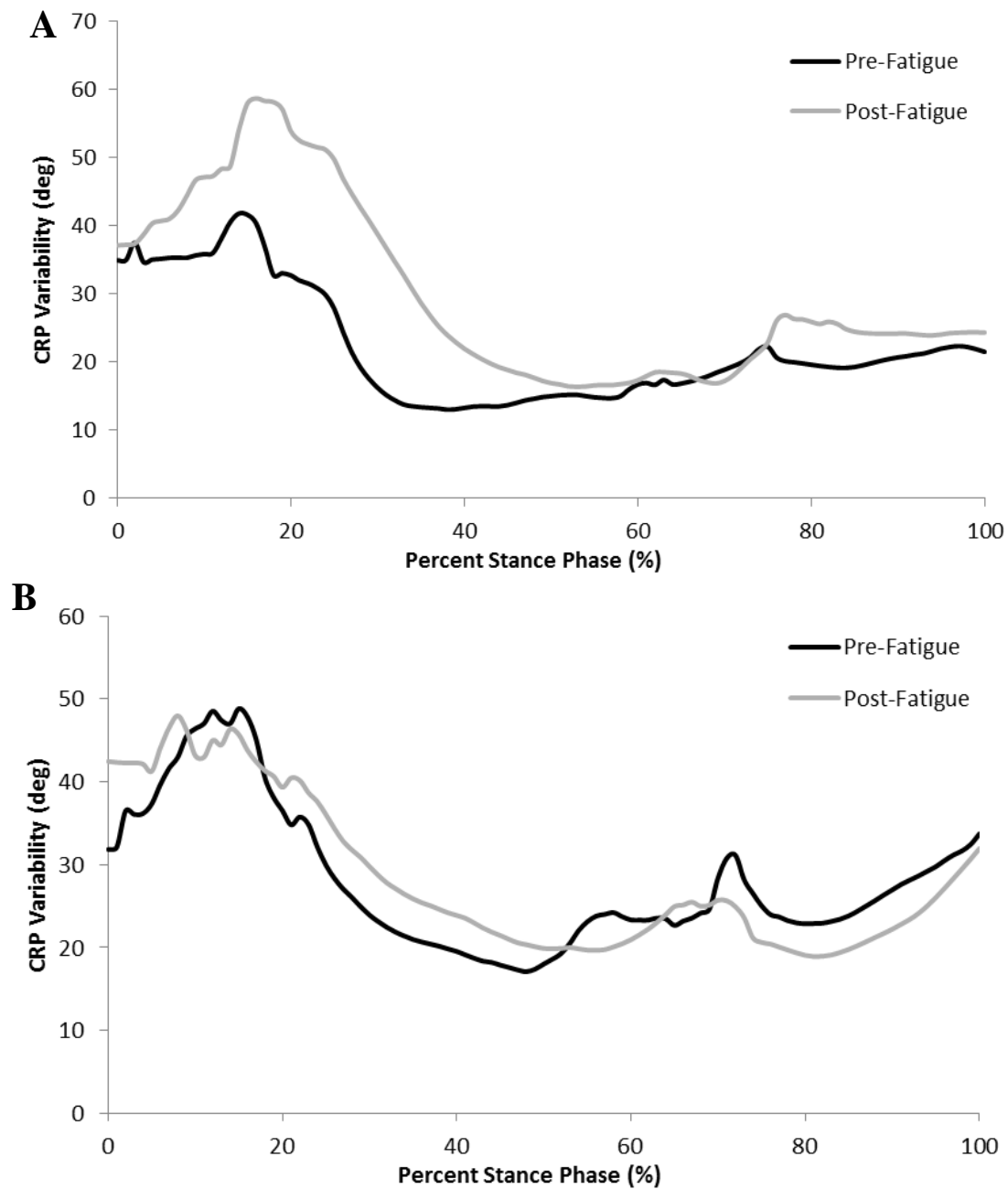


Figure 12. Frontal plane CRP variability before and after fatigue in **A)** females and **B)** males. CRP variability significantly increased after the fatigue protocol from 20-40% of stance phase, which was primarily driven by the overall increase in CRP variability in females.

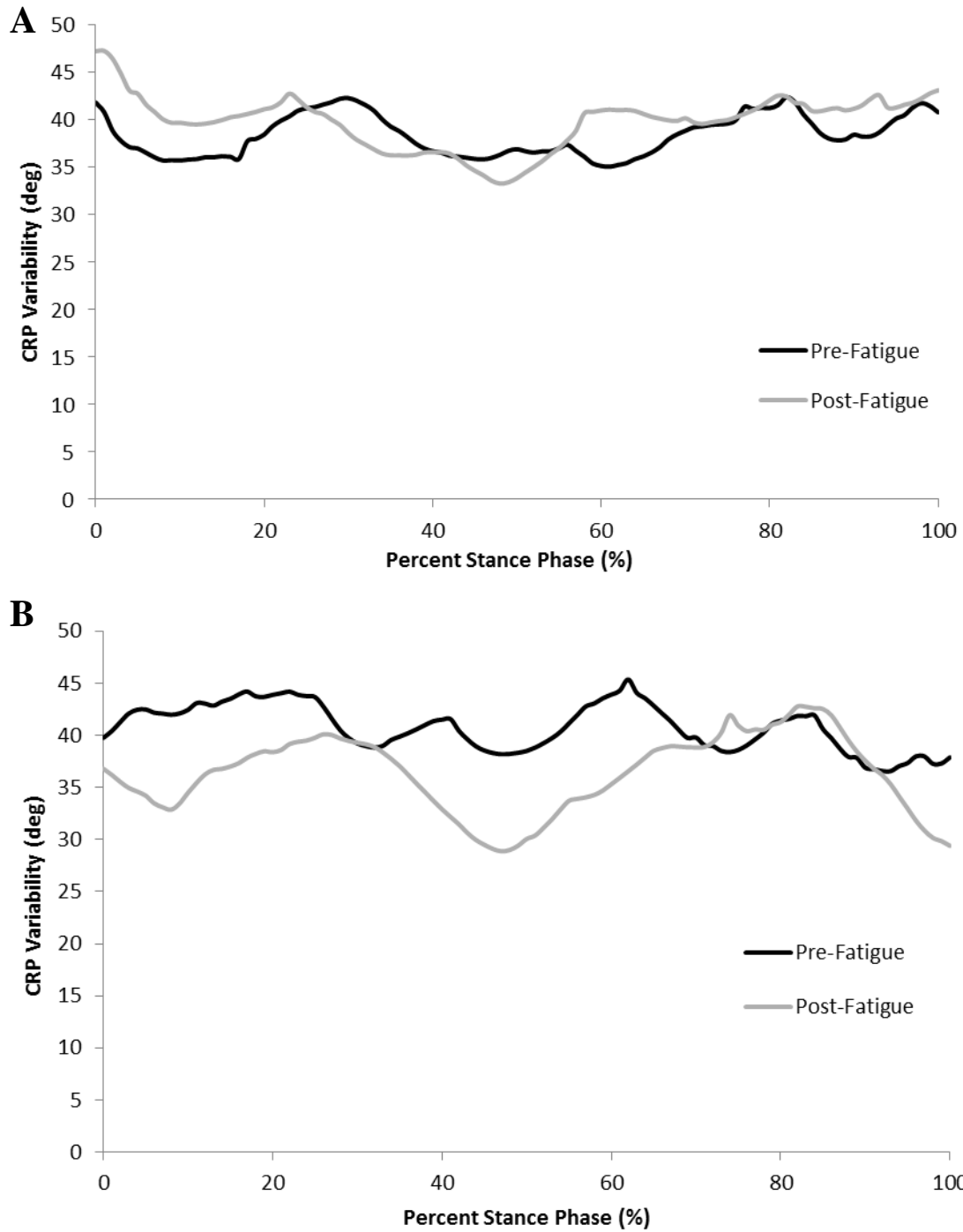


Figure 13. Transverse plane CRP variability before and after fatigue in **A)** females and **B)** males. There were no interaction effects or main effects for any variable.

In the pre-fatigue sex comparison, there was no interaction effect, nor main effect of sex for sagittal plane CRP variability. There was a main effect of time, $p < .001$ (Figure 14). In the frontal plane, there was no interaction effect, but there was a main effect of sex, with females displaying less CRP variability than males (males: $25.90 \pm 9.87^\circ$; females: $20.54 \pm 9.87^\circ$; $p = .040$), and main effect of time, $p < .001$ (Figure 15). In the transverse plane, there was no interaction effect, nor main effects for sex or time. The main effect of sex was trending towards significance, however, $p = .077$ (Figure 16). Based on visual inspection of the data, a post-hoc analysis was performed on only the first 50% of stance phase. This analysis did find a main effect of sex, with females displaying significantly less variability compared to males from 0-50% of stance phase (males: 41.26 ± 15.26 ; females: $32.82 \pm 15.26^\circ$; $p = .036$).

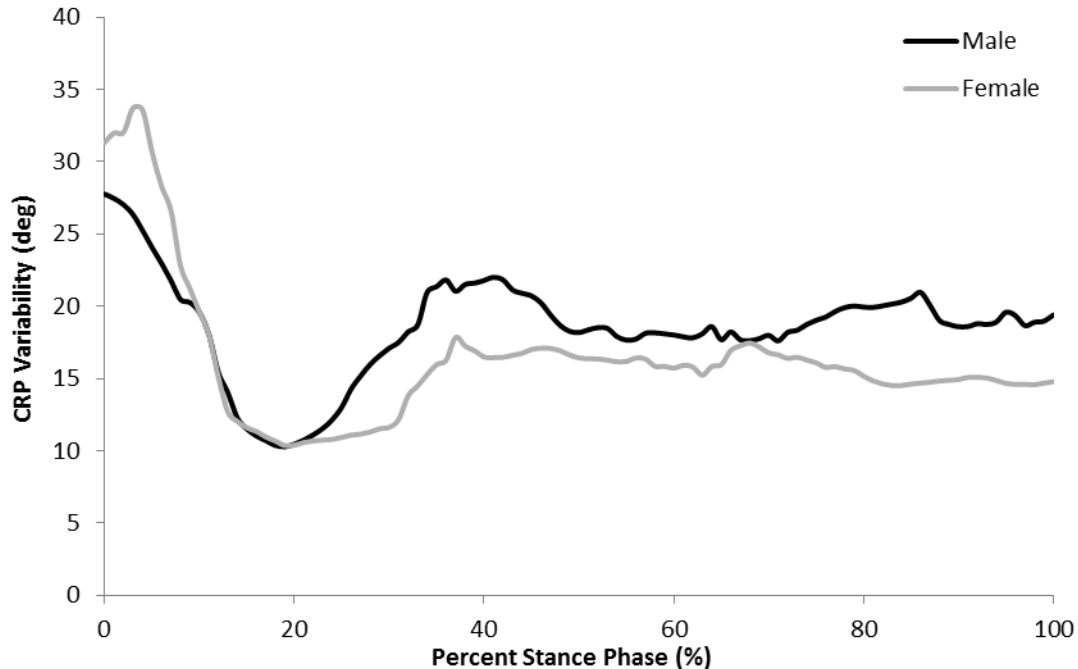


Figure 14. Sagittal plane CRP variability between males and females. There was no interaction effect or main effect of sex.

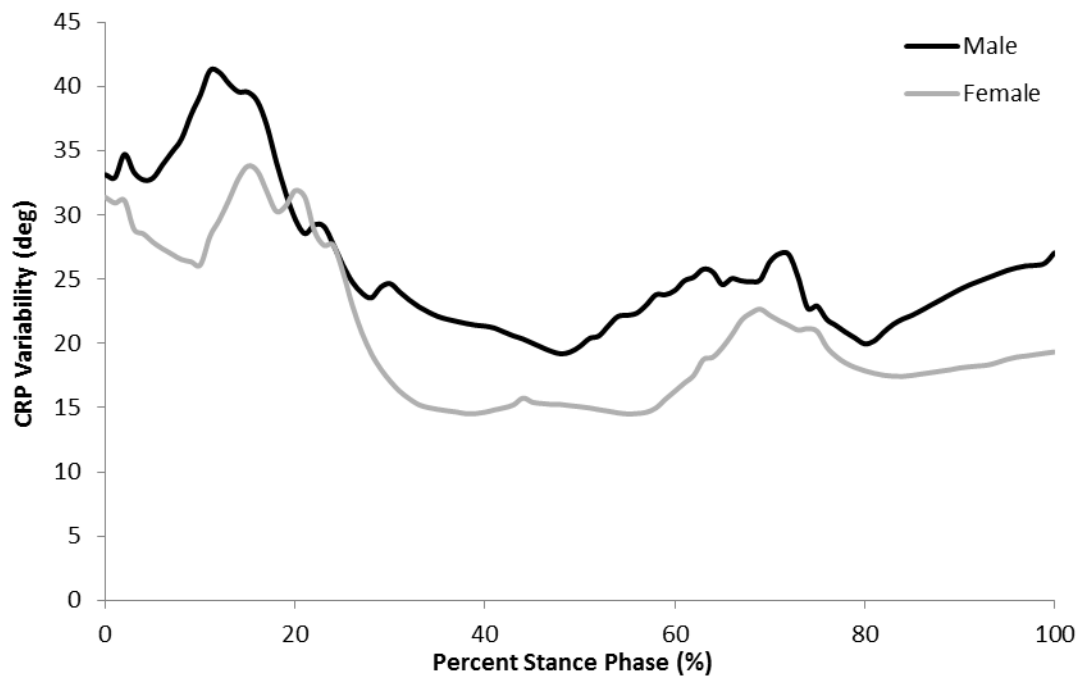


Figure 15. Frontal plane CRP variability between males and females. There was a main effect of sex, with females displaying less CRP variability, $p = .040$.

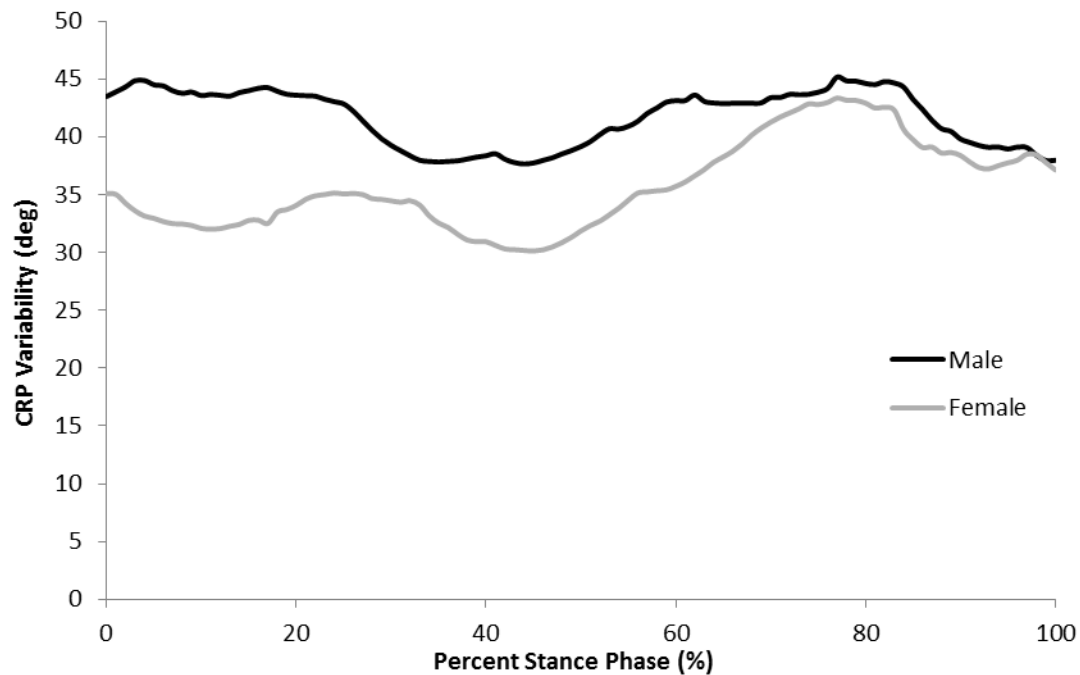


Figure 16. Transverse plane CRP variability between males and females. There was no interaction effect, and the main effect of sex was trending towards significance, $p = .077$. A post-hoc analysis indicated that females displayed significantly less CRP variability during the first 50% of stance phase, $p = .036$.

Discussion

One of the main purposes of this study was to investigate the effect of decreasing hip abduction strength on CRP variability at the hip. Decreasing hip abduction strength did not significantly affect CRP variability in either the sagittal or transverse planes. In the frontal plane, decreasing hip abduction strength significantly increased CRP variability from 20-40% of stance phase, which appears to be primarily driven an overall increase in CRP variability in females.

Greater coordinative variability, as seen in females after the fatigue protocol, is generally thought to be injury-protective, as it may help distribute joint loading across a greater contact area (44). However, there may also be a threshold where coordinative variability is too high, as instability at a joint may predispose an individual to injury (44). Decreased hip abduction strength likely increases instability at the hip joint, which led to greater CRP variability in females. Interestingly, average CRP variability was higher in females after the fatigue protocol ($29.92 \pm 11.85^\circ$) than in males before the fatigue protocol ($25.90 \pm 9.87^\circ$). However, the exact threshold for excessive CRP variability is unclear (44), so the results from this study cannot determine whether decreased hip abduction strength may increase or decrease injury risk in females.

Comparing CRP variability between sexes before the fatigue protocol, females demonstrated less variability than males in the frontal plane. Previous research suggests that healthy female runners run with greater peak hip adduction during running compared to healthy males (31), which may decrease contact area and increase contact stress on the lateral patella (50) as well as increase the external knee abduction moment (103). Low frontal plane hip coordinative variability during midstance could thus elevate lateral

patellar contact stress even more by further decreasing the surface area available to distribute these high internal forces (44).

While not significant across the entire stance phase, females demonstrated greater CRP variability than males over the first 50% of stance phase. This represents the loading phase of running, where impact forces rapidly increase joint stress, and muscular forces at the hip must decelerate the body's center of mass and control hip internal rotation (102). Healthy females, on average, run with greater hip internal rotation during running compared to males (31), which can increase lateral contact stress on the patella, especially if femoral rotation exceeds 20° (62). Similar to the frontal plane, less CRP variability in the transverse plane in females may further increase contact stress on the lateral patella.

This study was not without a few limitations. Running speed was not tightly controlled, as all subjects ran at their self-selected easy pace. Running speed was not different between males and females (Chapter II, Table 1), but there was a small but significant increase in running speed after the fatigue protocol. This change in running speed was controlled for as a covariate in the analysis. Foot strike patterns were not controlled in this study, so it is not clear if coordinative variability at the hip is affected by the type of foot strike. Running shoes were also not standardized in this study, so that coordinative variability was not affected by novel footwear. CRP variability between different planes of motion was not analyzed in this study (i.e. transverse plane pelvis motion and frontal plane hip motion), which has been used to quantify coordination between distal segments during running (42). Finally, this study did not look at

coordinative variability at the knee, which is the next logical step given that the knee is the actual site of injury in PFPS.

In conclusion, decreasing hip abduction strength increased frontal plane CRP variability at the hip from 20-40% of stance phase, primarily in females. It is unclear whether this increase exceeds the threshold for excessive coordinative variability that may be indicative of injury. Females demonstrated less CRP variability at the hip than males in the frontal plane, and during the first 50% of stance phase in the transverse plane, which may have implications for knee injury.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Summary of Main Findings

The overall aim of this study was to better understand the relationships between hip muscle strength, hip muscle activity, and running kinematics, particularly at the hip. Chapter II specifically looked at relationship between isometric hip muscle strength and hip muscle activation during running in both males and females. Overall, hip strength and hip muscle activation during running were not strongly correlated, as the only significant correlation found was between hip abduction strength and TFL onset in females. Because the TFL does not provide is not a large contributor to hip joint stability (65), this one finding likely does not have a substantial clinical implication. Thus, the hip strength measurements used in clinical settings appear to not directly affect the amplitude or timing of hip muscle activation during running.

This study also found that males displayed greater isometric hip flexion, internal rotation, and external rotation strength compared to females. Importantly, no significant differences were seen in hip abduction strength between sexes. This finding is in direct disagreement with many published studies, but does agree with previous findings from this laboratory (46). In addition, females displayed delayed and shorter gluteus maximus activation, but greater relative amplitude compared to males. Delayed gluteus maximus onset has been correlated to greater hip adduction and hip internal rotation excursion in females with PFPS (117). Also, greater relative GMAX peak amplitude may lead to greater muscle fatigue and thus poorer dynamic control of the hip during prolonged

running (116). Thus, these finding may help explain a one reason for the female sex bias in PFPS.

The second study used a multiple regression approach to look for predictors of hip adduction and hip internal rotation during running. For hip adduction excursion, sex, running speed, static subtalar inversion, and gluteus maximus amplitude were significant predictors in the model, with females displaying significantly greater hip adduction excursion compared to males. GMAX amplitude's inclusion in the model is interesting given the trend towards greater gluteus maximus activation in females from Chapter II. Based on the combined results of these studies, female sex, gluteus maximus activation, and hip adduction excursion all appear to be related. It was hypothesized that runners with excessive hip adduction (as seen in more often in females) activate gluteus maximus at a higher percent of the maximum in an attempt to control frontal plane hip motion, meaning GMAX amplitude is less of a “predictor” and more of a “result” of greater hip adduction excursion. This hypothesis, however, is purely speculation.

The inclusion of static ankle inversion range of motion is a novel finding. While it was hypothesized that greater available subtalar ROM may increase the available range of motion for hip adduction during running, this idea is also purely speculative and needs further exploration.

Unfortunately, the only significant predictor in the hip internal rotation excursion model was sex, as females displayed significantly greater hip internal rotation excursion compared to males. Thus, more work needs to be performed to determine any factors which may contribute to hip internal rotation excursion during running.

The third study examined the effect of decreasing hip abduction strength on

running kinematics and hip muscle EMG. Neither hip kinematics nor gluteus medius activation changed after the fatigue protocol, which may help explain why hip strengthening programs do not change hip kinematics during running. However, we did see some changes in kinematics, most notably at the trunk, with increased ipsilateral lean. This kinematic compensation is likely an attempt to offload the hip abductors, and may have a deleterious effect on the knee joint moment (37).

In addition, gluteus maximus peak amplitude and duration increased after fatigue, as did TFL peak amplitude. Assuming that the dynamic control of the hip joint was hindered after the fatigue protocol, this finding further supports the notion that gluteus maximus activity increases to help control the thigh, especially in the frontal plane.

Finally, the fourth study used an alternative biomechanical method called continuous relative phase (CRP) to look the effect of sex and decreased hip abduction strength on CRP variability. Decreasing hip abduction strength increased frontal plane CRP variability at the hip from 20-40% of stance phase, primarily in females. It is currently unclear if this increase may exceed a threshold that increases injury risk in females. Before the fatigue protocol, females demonstrated less CRP variability at the hip than males in the frontal plane and transverse planes. Due to the relationship between hip adduction, hip internal rotation, and PFPS risk, decreased CRP variability may have profound injury implications and help explain the sex bias for PFPS.

Clinical Implications

Overall, the results from this study shed light on the relationships between hip strength, hip muscle activation, and kinematics during running in both males and females,

which may have implications for injury and rehabilitation strategies.

First, hip muscle strength does not appear to be strongly correlated with hip muscle activation or hip kinematics during running. Thus, if the goal of rehabilitation is to decrease “excessive” hip adduction, hip strengthening alone will likely be ineffective, and methods of gait retraining may be indicated. However, hip strength may be an important modulator of trunk motion, which can have a substantial impact on knee joint loading (28,64). Thus, hip muscle strengthening should still be an important component of rehabilitation for knee injuries.

Gluteus maximus amplitude and timing appear to be different between males and females during running. In addition, gluteus maximus amplitude appears to be related to hip adduction excursion, and may have an inverse relationship with hip abduction strength. While most research has focused on hip abduction strength and gluteus medius function during running, gluteus maximus appears to be important in providing stability to the hip, not just forward propulsion, and may require increased attention during rehabilitation. One method clinicians may utilize is increasing cadence, which has previously been shown to decrease gluteus maximus peak amplitude during (65). Also, increasing hip extension strength may be warranted due to the trending correlation between hip extension strength and GMAX onset seen in Chapter II.

Finally, lower coordinative variability at the hip was seen in females in the frontal and transverse planes compared to males, which may have injury implications. While coordinative variability appears to be a very useful biomechanical tool, its ability to enhance clinical decisions is currently limited. Thus, efforts need to be made to help bridge the gap between biomechanics and clinical application for this type of analysis.

Directions for Future Research

This entire dissertation was comprised of data on healthy runners. Thus, most of the discussion was centered around factors and relationships that “may have implications for injury”, or “may help explain the female sex bias for injury”. The next logical step is to perform similar experiments on a cohort of males and females with patellofemoral pain. Specifically, this future research could look at whether runners with PFPS display different relationships between the variables studied, demonstrate different compensations to decreased hip abduction strength, have lower coordinative variability at the hip compared to their healthy counterparts, or fatigue to a greater extent.

Other biomechanical techniques could be used to analyze this data. Knee joint forces and moments were not analyzed in this study, but do have a significant relationship to knee injury. In particular, it would be very interesting to quantify the effect of decreased hip abduction strength on knee joint moments. Also, using musculoskeletal modeling software like OpenSim could also be used to estimate the effect of decreased hip abduction strength on hip muscle forces.

This dissertation mostly ignored the contribution of foot, ankle, and shank mechanics to the relationships studied. It also only sampled EMG data from a very small subset of muscles. Future studies could add in more distal contributions and EMG measures into similar comparisons.

Hopefully, the combination of findings from this dissertation, contributions from other researchers, and the future directions suggested will help clinicians improve the care and rehabilitation practices of runners suffering from patellofemoral pain syndrome.

APPENDIX A

DATA COLLECTION FORMS

Running Study Subject Questionnaire and Clinical Evaluation Form

Subject Code: _____ Date: _____

Sex: _____

Age: _____

Number of Years Running: _____

Approximate Mileage Run per Week: _____

During your running career, have you sustained any running related injuries? **Yes/No**

If **Yes**, then please describe the nature of the injury, diagnosis by a physician, extent or duration of the injury, and treatment protocols you underwent to relieve symptoms:

Other Comments of History Information:

Subject #: _____

<u>Parameter</u>	<u>Shod</u>	
	L	R
Height (in cm)		
Body Mass (kg.)		
ASIS Width (cm)		
Thigh Length (cm)		
Mid-Thigh Circumference (cm)		
Calf Length (cm)		
Calf Circumference (cm)		
Knee Diameter (cm)		
Malleolus height (cm)		
Malleolus width (cm)		
Foot Length (cm)		
Foot Width (cm)		

General Lower Body Alignment and Mobility Assessment

1. Standing Leg Varus to Floor
 - a. Left: _____
 - b. Right: _____
2. Standing Arch Type:
 - a. Left: _____
 - b. Right: _____
3. Stork Test (normal or restricted)
 - a. Left: _____
 - b. Right: _____
4. Trendelenburg Test
 - a. Left: _____
 - b. Right: _____
5. Ankle Dorsiflexion

	Extended	Flexed
Left		
Right		
6. Ankle Plantarflexion (flexed)
 - a. Left: _____
 - b. Right: _____
7. Subtalar Joint Inversion
 - a. Left: _____
 - b. Right: _____
8. Subtalar Joint Eversion
 - a. Left: _____
 - b. Right: _____
9. Forefoot Alignment (Neutral, Varus, Valgus)
 - a. Left: _____
 - b. Right: _____
10. 1st MPJ ROM (dorsiflexion)
 - a. Left: _____
 - b. Right: _____
11. Prone Hip Internal Rotation
 - a. Left: _____
 - b. Right: _____
12. Prone Hip External Rotation
 - a. Left: _____
 - b. Right: _____
13. Quadriceps Flexibility
 - a. Left: _____
 - b. Right: _____
14. Extremity Length (cm)
 - a. Left: _____
 - b. Right: _____
15. Hamstring 90-90 Test
 - a. Left: _____
 - b. Right: _____
16. Thomas Test
 - a. Left: _____
 - b. Right: _____
17. Ober's Test
 - a. Left: _____
 - b. Right: _____
18. Tibial Torsion
 - a. Left: _____
 - b. Right: _____

Measurement of Arch Height

(from Williams, McClay, Hammill, and Buchanan (2001). Lower Extremity Kinetic and Kinematic Differences in High and Low Arched Runners. *J. Applied Biomechanics*. Vol. 17, pp. 153-161)

1. Full Foot Length (cm): L _____ R _____

2. 50% Full foot length (cm): L _____ R _____

3. Truncated Foot Length _____ L _____ R _____

(measured from most posterior point of calcaneus to medial joint space of first metatarsal phalangeal joint).

4. Height of Dorsum of foot @ 50% foot length: _____ L _____ R _____

5. Arch Height Ratio: _____ L _____ R _____

(measurement 3 divided by measurement 4)

Clinician Notes/Comments:

APPENDIX B

INFORMED CONSENT FORM

You are invited to participate in a research study conducted by doctoral candidate JJ Hannigan, MS, ATC and Drs. Li-Shan Chou, Louis Osternig, Stan James regarding the effect of hip muscle fatigue on hip muscle activity and running mechanics. You are being invited to participate because you are currently 18-45 years old and running greater than 20 miles per week.

TESTING PROCEDURES: If you decide to participate, you will visit the Motion Analysis Laboratory at the University of Oregon for 2-3 testing sessions. During visit 1, body measurements, including height, weight, and leg length, will be measured. You will then undergo a clinical exam assessing general lower limb alignment, flexibility, and joint mobility, which will take approximately 45 minutes to complete. Next, we will test your hip muscle strength on a machine called a Biodex dynamometer, which measures how much muscle force you produce in a given direction. You will be asked to push against the dynamometer in 6 different directions per leg. These tests will take approximately 45 minutes, totaling ~1.5 hours for visit 1.

During Visit 2, after cleaning the skin with an alcohol solution, you will be fitted with 6 pairs of active surface electrodes over 3 hip muscles in each leg, as well as 1 ground electrode. These surface electrodes are tiny sensors that can detect the electrical activity of your muscles. Hypo-allergenic conducting gel will be placed between the sensor and your skin to improve conductivity. You will then be asked to perform a series of maximum voluntary isometric contraction (MVC) attempts for both hip abduction (pushing out to the side) and hip extension (pushing backwards) using the Biodex dynamometer.

You will then be outfitted with 39 reflective markers and run overground for approximately 10 minutes while your motion is recorded using the lab's motion capture system, which uses special 3D cameras to track your motion. After 10 minutes, you will undergo a hip abductor fatigue protocol, which will involve maximally pushing against a resistance in the direction of abduction (pushing out to the side). The resistance will rotate at a constant speed up and down while you maintain contracting in the direction of abduction. This test will be stopped after 2 minutes. You will immediately perform an MVC and then run again for approximately 10 minutes. In total, visit 2 will take approximately 2.5 hours.

If you decide to come back for visit 3, you will again be outfitted with 39 reflective markers. We will test your hip abduction strength, have you run for approximately 30 minutes at around your 5k running pace, and then immediately test hip abduction strength again. This will take approximately 1 hour.

COMPENSATION: You will be compensated \$10 per hour, for a maximum total of \$50 if you complete all 3 visits. Visit 1 = \$15, Visit 2 = \$25, and Visit 3 = \$10.

RISKS AND DISCOMFORTS: We expect that there will be no more risk for you during these tests than there normally is for you when outside of the laboratory. Running in the laboratory may be slightly different than running outside requiring them to speed up and slow down quickly as well as navigate tight corners. While these conditions should not pose any risks or hazards, care will be taken to allow you to take breaks or cease data collection if needed.

In order to place electrodes on your hip muscles, we will need to partially expose your buttocks and lateral thigh in order to properly palpate these muscles to place the electrodes on your skin. This will be done in a separate room with the curtain pulled. A researcher of the same sex as you will place the electrodes. We realize that these areas can be sensitive for some subjects. If you are uncomfortable with partially exposing these areas for electrode placement, it is probably best to not participate in this study.

Occasionally, an individual may feel light-headed or dizzy from smelling the alcohol solution used to clean your skin. If you feel light-headed at any time, please notify the investigators so that we can stop the procedure. Additionally, the hypo-allergenic conducting gel can produce an allergic response, such as redness or itching, in some subjects. If this occurs, it will be limited to the region of skin the gel is applied. If you experience undue discomfort from this gel, please again notify the investigators.

During the fatigue protocol, you will likely feel some discomfort, specifically in the muscles being fatigued. This discomfort is expected and normal. Additionally, some subjects may experience fatigue and/or soreness in the hip abductor muscles for up to 72 hours following the test. Please let us know if you experience excessive discomfort or any level of pain throughout the study. You are free to stop the test at any time without penalty for any reason.

ADDITIONAL INFORMATION: Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will not be shared without your permission.

Subject identities will be kept confidential by coding the data as to study, subject pseudonyms, and collection date. The code list will be kept separate and secure from the actual data files.

Your participation is completely voluntary. Your decision whether or not to participate will not affect your relationship with the Department of Human Physiology or University of Oregon. You do not waive any liability rights for personal injury by signing this form. In spite of all precautions, you might develop medical complications from participating in this study. If such complications arise, the researchers will assist you in obtaining appropriate medical treatment. In addition, if you are physically injured because of the project, you and your insurance company will have to pay your doctor bills. If you are a University of Oregon student or employee and are covered by a University of Oregon medical plan, that plan might have terms that apply to your injury. If you have any questions about your rights as a research subject, you can contact Research Compliance Services, 5237 University of

Oregon, Eugene, OR 97403, (541) 346- 2510. This office oversees the review of the research to protect your rights and is not involved with this study.

If you decide to participate, you are free to withdraw your consent and discontinue participation at any time without penalty.

If you have any questions, please feel free to contact JJ Hannigan, (541) 346-1033 or Dr. Li-Shan Chou, (541) 346-3391, Department of Human Physiology, 112C Esslinger Hall, University of Oregon, Eugene OR, 97403-1240. You will be given a copy of this form to keep. Your signature indicates that you have read and understand the information provided above, that you willingly agree to participate, that you may withdraw your consent at any time and discontinue participation without penalty, that you will receive a copy of this form, and that you are not waiving any legal claims, rights or remedies.

Name: _____

Signature: _____

Date: _____

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